Intracardiac
Phenomena

This work was done during the tenure of a Teaching Grant of the National Heart Institute, U S P H.S by Dr Luisada

INTRACARDIAC PHENOMENA

In Right and Left Heart Catheterization

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A Second Edition Revised and Enlarged, of

CARDIAC PRESSURES AND PULSES



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Introduction to the Second Edition

Part of the material presented here was published in 1956 under the title "Cardiac Pressures and Pulses." A revision was imposed by the development of left heart catheterization, due to Bjoerk, Kent and Fisher, and Morrow. As this method has been applied in our laboratory since 1956, we are now able to present original documents.

Intracardiac phonocardiograms and electrocardiograms, recorded in this laboratory for over two years, cannot be separated from pressure tracings in their interpretation. Therefore, the scope of this book has been extended to include the results obtained through use of these methods.

Technical details, analysis of blood gases and "formulas" have been included simply in order to help the reader without obliging him to consult other works. Therefore this monograph should not be considered as a "manual" and does not pretend to be one.

The presentation of personal viewpoints will add, we hope, to the value of this monograph for those wishing to increase their knowledge in the fascinating field of "Intracardiac Phenomena."

The authors wish to acknowledge the work of the various members of their *cardiac team*, and particularly Drs. C. Aravanis, M. R. Testelli, J. Szatkowski and J. Bendezu as well as that of Mr. J. Morris and Mrs. S. Misic.

ALDO A. LUISADA
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Chicago 1958

Introduction to the First Edition

Catheterization of the right heart was initiated by a German surgeon (Forssmann) in 1930. It is not surprising that a surgeon, and not a physician was the first to try this technique. At that time, physicians were far less familiar with the use of catheters and intravenous drip methods.

While Forssmann's attempts on himself were considered at the time only an unusual technical exercise, Cournand and his group perfected this method to such a degree that it was gradually accepted and utilized by more and more researchers. Dexter, Lenègre, McMichael, P. Wood and Burchell, together with their teams, further developed the techniques which are currently used.

The method, as originally described, was based on the oxygen determination of blood samples aspirated from the chambers of the right heart or pulmonary artery, and on pressure measurements. Later catheterization of the chambers of the left heart through septal openings added interesting data to those already known. Intracardiac electrocardiography, determination of cardiac output by means of the Fick principle, and study of the pulmonary venous pressure by firmly wedging the catheter in a pulmonary arteriole were subsequent additions.

Left heart catheterization is a relatively recent development which will very likely extend the realm of this technique to include a much larger group of patients. Cases with aortic valve lesions (which are outside the realm of right heart catheterization) and cases with mitral valve lesions (in which right heart catheterization gives only indirect data) may be studied by left heart catheterization.

Pressure tracings are usually taken with a conventional film speed which is inadequate for the study of accurate details of the pulse. This small monograph is based on tracings recorded with a somewhat different technique, aiming to study in detail various patterns of the pressure pulses of the heart and vessels.

Cardiovascular physiology, details of technique, formulas used in catheterization and a study of artifacts have been included in this volume to simplify the task of young cardiologists who may wish to begin working in the fascinating and ever expanding field of cardiac catheterization.

The authors wish to thank Dr. A. B. Lima, who collaborated in the study of some cases during his sojourn in Chicago in 1953–1954.

ALDO A. LUISADA

CHAPTER ONE

The Cardiac Cycle

CONTRACTION OF THE ATRIA

The cardiac cycle starts with *the contraction of the atria*, also called "atrial systole" A wave of contraction follows that of excitation, moving downward from above thus creating a propulsive wave toward the ventricles An appreciable backflow toward the large veins is prevented by the initial contraction of the musculature at the opening of the veins and by the high venous pressure accompanying this phase

Atrial contraction takes place during that short phase which immediately precedes ventricular contraction the *presystole* As the A V valves are open during atrial contraction, only a moderate rise in pressure takes place within the atria and the contraction is mainly revealed by movement of blood (Fig 1)

Atrial contraction is not indispensable for ventricular filling because most of it occurs during early diastole Still contraction of the atria completes ventricular filling and is one of the factors upon which the normal function of the A V valves is based In rapid heart action and in mitral stenosis atrial contraction acquires a much greater importance

CONTRACTION OF THE VENTRICLES

Initiation of ventricular contraction increases the pressure in the ventricles and closes the *atrioventricular valves* (tricuspid valve in the right heart mitral valve in the left heart) Immediately afterwards, the contraction of the papillary muscles prevents an eversion of these valves and permits a further rise of pressure to a point equaling and then exceeding the pressures existing in the aorta and the pulmonary artery In this short period the ventricular contraction builds up pressure without causing motion of blood This short phase is called *period of tension or period of isometric contraction* because the muscle fibers of the ventricles build up tension steadily even though unable to become shorter

At the onset of ventricular contraction the entire myocardium has been excited Owing to the latency between excitation and contraction, however those fibers which were excited first start contracting first Then more and more fibers contract For this reason ventricular pressure rises slowly at first, then very rapidly

As soon as the ventricular pressure exceeds that of the respective artery *the semilunar valves open* and *the outflow begins* During this period

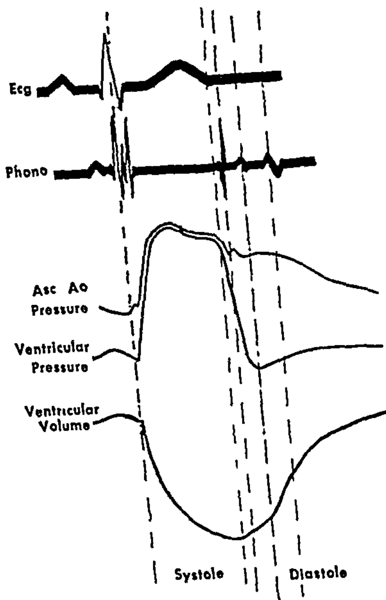


Fig 2—Changes of ventricular pressure and volume and pressure in the ascending aorta during the cardiac cycle correlation with heart sounds (phono) and electrocardiogram (ECG)

merely touch but form a surface contact without folds Closure is started by the eddy currents and is increased by the ventricular contraction which immediately follows Eversion is prevented by the chordae tendineae held by the papillary muscles The musculature of the septum and of the papillary muscles is the first to contract, insuring a timely closure of the valves The termination of atrial contraction contributes to the closure of the A V valves because the leaflets are brought into

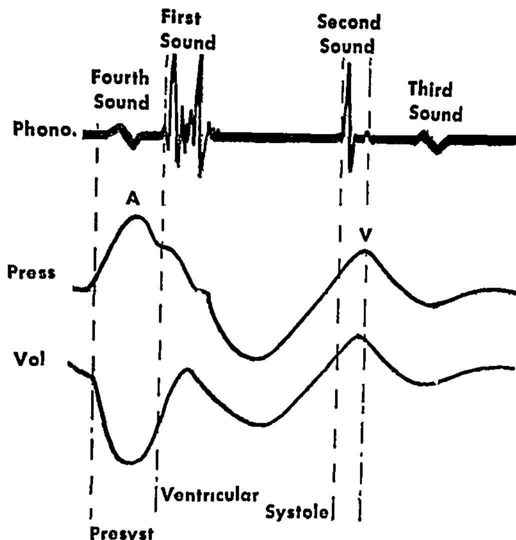


Fig 1—Changes of atrial pressure (press) and volume (vol) during the cardiac cycle and correlation with heart sounds tracing (phono)

of *outflow* or *ejection*, the fibrous septum which supports the A V valves is lowered by the contraction of the ventricles (papillary muscles, free ventricular wall, and intraventricular septum) As a consequence, a remarkable increase in size of the atria takes place during systole, causing suction of blood from the veins (Fig 5)

From beginning to end, the ventricular pressure follows a steady course During the last part of ejection, outflow is decreased Therefore ejection has been divided into two parts *maximum ejection*, which includes about one half of the time, and during which the ventricle expels about two thirds of the blood, and *reduced ejection* during which the ventricle expels about one third of the blood in the last half of the time

MOVEMENTS OF THE VALVES

Despite their apparently delicate structure, the *flaps of the A V valves* have considerable strength and resistance When closed, they do not

(Fig 3 B) Each peak of rapid filling follows A V valve opening by 0.08 to 0.10 sec and the closure of the respective semilunar valve by 0.12 to 0.18 sec

Table I shows the sequence of events in normal large dogs whose figures are practically identical with those of normal man

CHANGES OF CARDIAC DIAMETERS

During ventricular contraction, *all diameters of the heart decrease* the base is pulled downward and the large vessels are stretched while the apex does not move upward (Fig 5) The spiral arrangement of the muscular bundles of the ventricles makes their contraction very efficient so that the blood is virtually wrung out It also causes the heart to rotate to the right pressing the apex more firmly against the chest wall This, together with the increased firmness of the ventricular mass, is the cause of the so called *apex beat* In spite of the progressive contraction of the free ventricular walls and of the septum, a small amount of blood is left within the ventricles even in normal conditions (*residual blood*)

The interventricular septum seems to have an important dynamic role, a large part of right ventricular ejection seems to be due to this structure

VENTRICULAR DIASTOLE

At the end of ventricular systole ventricular pressure drops to zero Following an extremely brief interval of latency (so called *protodiastole*) the semilunar valves of the aorta and pulmonary artery close A short

TABLE I *Time Intervals Between Valvular Motions (Normal Dogs)*

Event	Interval (a second)	Symbol	Heart Sound
Q wave (ECG)			
	0.06-0.07		
Closure of mitral valve		M	
	0.00-0.02		
Closure of tricuspid valve		T	First Sound — major vibrations 0.06-0.08
	0.01-0.03		
Opening of pulmonic valve		P	
	0.01-0.02		
Opening of aortic valve		A	
Closure of aortic valve		A	
	0.02		
Closure of pulmonic valve		P	
	0.03-0.04		Second Sound — major vibrations 0.04-0.05
Opening of tricuspid valve		OT	
	0.04-0.08		
Opening of mitral valve		OM	
	0 -0.04		
Rapid filling of right ventricle		3R	
	0.04-0.08		Third Sound
Rapid filling of left ventricle		3L	

position by the eddy currents set up by the flow through the orifices and by a reversal of the pressure gradient. This is shown by the temporary valvular insufficiency which frequently develops in cases with incomplete A-V block. In the event of delayed A-V conduction, there may be a double closure of the A-V valves: the first at the end of atrial contraction, the second at the beginning of ventricular systole.

Since the efflux of blood from the branches of the aorta is faster than ejection from the left ventricle, the pressure gradually declines in the aorta during the second part of systole. This drop of aortic pressure may be responsible for a slight drop of pressure in the ventricular curve (Fig 2).

The *semilunar valves of the aorta and pulmonary artery* resemble pockets attached to the wall of the vessel. The blood contained in the pockets keeps the valves away from the wall. Both the reversal of the gradient of pressure created by the sudden cessation of outflow and the eddy currents cause closure of these valves at the end of ventricular systole. Firm attachment of the valves, muscular support from the ventricular base, and lateral apposition prevent any possibility of eversion, in spite of the lack of chordae tendineae.

Research in normal animals by means of multiple heart catheterizations has revealed the following data (Luisada et al, 1958):

(a) The interval between beginning of rise of pressure in a ventricle and the crossing of the atrial pressure curve is extremely short (in the range of 0 01 sec or less) (Fig 3 A). This indicates that closure of the mitral and tricuspid valves occurs practically at the onset of ventricular systole.

(b) In certain of the cycles *left* ventricular pressure rises first and increases at a faster rate. *Right* ventricular pressure lags behind by 0 01 to 0 02 sec and rises more slowly (Fig 3 B). It is reasonable to suppose that in these cycles mitral closure precedes tricuspid closure by 0 01 to 0 02 sec. This is confirmed by external and intracardiac phonocardiography (see below). In other cycles the two pressures rise simultaneously, the difference often shown by two subsequent cycles (Fig 4 A). It is likely that in those cycles mitral and tricuspid closures occur *simultaneously*.

(c) The rise of pressure in the pulmonary artery always precedes that of the aorta. The interval is usually in the range of 0 02 sec and may become longer or shorter if there is a marked change of pressure in one of the vessels (Fig 3 B).

(d) The relationship of mitral closure to aortic opening and of tricuspid closure to pulmonic opening is such that the following isometric periods should be accepted:

(1) RV—0 02–0 04 sec
(2) LV—0 05–0 06 sec

Thus the following sequence of events takes place:

(1) Mitral closure
(2) Tricuspid closure
(3) Pulmonic opening
(4) Aortic opening

(e) The end of *left* ventricular systole always *precedes* that of the right (Fig 3 B).

(f) The *opening* of the mitral valve always *follows* that of the tricuspid. This interval is usually between 0 04 and 0 06 sec but may reach even 0 08 sec (Fig 3 B).

(g) Rapid filling of the *left* ventricle always *follows* that of the right. The interval between the two is similar to that occurring between the opening of the A-V valves

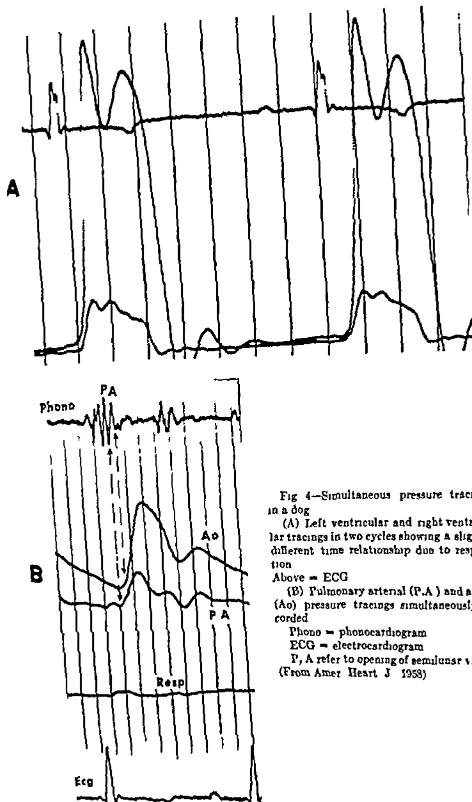


Fig 4—Simultaneous pressure tracings in a dog

(A) Left ventricular and right ventricular tracings in two cycles showing a slightly different time relationship due to respiration

Above = ECG

(B) Pulmonary arterial (P.A) and aortic (Ao) pressure tracings simultaneously recorded

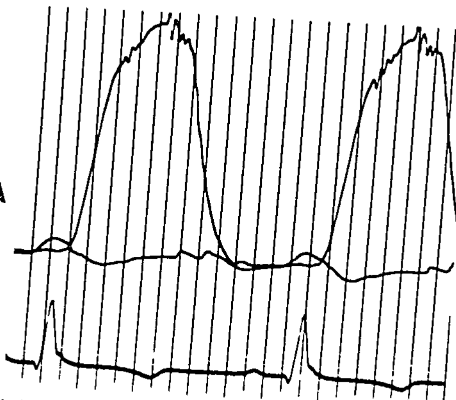
Phono = phonocardiogram

ECG = electrocardiogram

P, A refer to opening of semilunar valves

(From Amer Heart J 1958)

A



B

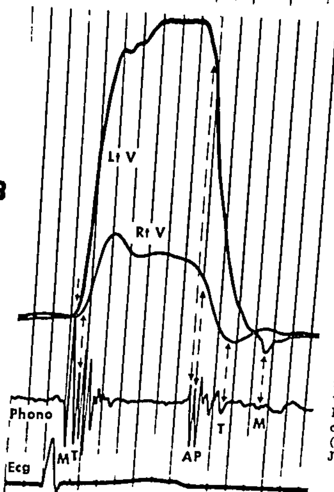


Fig 3—Intracardiac pressure tracings in a dog (closed chest)

(A) Left ventricular and left atrial pressures simultaneously recorded Below = ECG

(B) Left ventricular (Lt V) and right ventricular (Rt V) pressure tracings simultaneously recorded

Phono = phonocardiogram recorded at apex M T A P refer to motions of the four valves

ECG = electrocardiogram

(From Amer Heart J 1958)

pressure rise takes place in the venoatrial reservoir as well as in the ventricles

(c) A late phase of *rapid active filling (presystole)* caused by the atrial contraction which completes ventricular filling As soon as the atrial contraction is completed the ventricles start contracting, because the descending stimulus has already reached the ventricular myocardium

It should be kept in mind that, during ventricular diastole the atrium and ventricle of each side of the heart form like a single chamber

DURATION OF CARDIAC PHASES

Certain time intervals may be considered typical of a normal heart (Table II)

Because of the thinness of the *right* atrial wall and its distensibility, the filling volume of this chamber is about twice that of the *left* atrium

As atrial contraction lasts but a small fraction of the total cycle (less than one tenth of a second) the atrial wall is relaxed during most of ventricular diastole and during all of ventricular systole Thus the atria act as reservoirs for the blood coming to the heart

The traction developed by the ventricular muscles and septum on the atrioventricular junction during systole dilates the atria by causing a phenomenon of suction This is rapidly transmitted to the venous system and accelerates the flow of blood toward the atria (Fig 1) This is proven not only by physiologic experiments but also by the fact that in clinical tracings of normal subjects recorded from the *veins or atria* the pressure drops during ventricular systole

The *atrial appendages* seem to have little propulsive function and serve as complementary spaces which fill the deep niches at the base of the heart during ventricular systole

In abnormal conditions as in cases with rapid heart rate, diastole shortens tremendously In such cases atrial contraction may include most or all of diastole and acquire much greater importance

THE HEART SOUNDS

Auscultation of the normal heart usually reveals two sounds (or *tones*), occasionally three Recording of the normal heart sounds by *means of* phonocardiography may reveal as many as four sounds

The *first heart sound* occurs at the beginning of ventricular systole and lasts through the tension period and the beginning of the ejection period The *second heart sound* is shorter it takes place at the end of systole during the phases of protodiastole and isometric relaxation The name "systolic sounds" has been suggested by one of the authors for these two constantly heard sounds The other two sounds less frequently heard take place during diastole The name "diastolic sounds" has been suggested for them

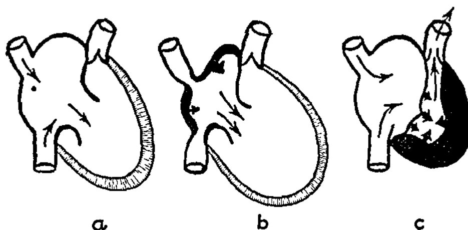


Fig 5—Schematic changes of cardiac shape during contraction (a) Mid diastole (free flow from veins to atria and ventricles) (b) Presystole (atrial contraction completing ventricular filling) (c) Ventricular contraction (expulsion of blood into the arteries lowering of A V floor, suction in the atria and veins)

time interval separates this phase from the subsequent opening of the A-V valves the *isometric relaxation period* Ventricular filling starts after the end of this period, e g, following the opening of the A V valves (Fig 2)

Filling of the ventricles has the following features

(a) An initial phase of *rapid passive filling* (*early diastole*) This is caused by the difference in pressure between the full atria and the empty ventricles At this time, the entire venoatrial reservoir experiences a drop in pressure due to acceleration of the stream after the opening of the atrioventricular valves Elastic recoil of the ventricular wall may accelerate filling (Brecher)

(b) A phase of *slow passive filling* (*mid diastole or diastasis*) The gradual filling of the ventricles slows down the inflow and a gradual

TABLE II *Time Intervals Between Cardiac Phases (Normal Dogs)* (in seconds)

	Right Heart	Left Heart
Ventricular Systole		
Tension	0 02–0 04	0 05–0 06
Ejection	0 18–0 28	0 17–0 26
Total Systole	0 20–0 32	0 22–0 32
Ventricular Diastole		
Protodiastole	0 02	0 04
Isometric relaxation	0 06–0 08	0 08–0 12
Rapid filling	0 08–0 10	0 08–0 10
Slow filling	variable	variable
Atrial dynamics	0 11–0 13	0 11–0 13

pressure rise takes place in the venoatrial reservoir as well as in the ventricles

(c) A late phase of *rapid active filling (presystole)* caused by the atrial contraction which completes ventricular filling As soon as the atrial contraction is completed, the ventricles start contracting because the descending stimulus has already reached the ventricular myocardium

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The following dynamic phenomena coincide with the heart sounds

Systolic Sounds

- **First sound**
  - Initiation of ventricular systole
  - Closing of the A-V valves
  - Opening of the semilunar valves
- **Second sound**
  - End of ventricular systole
  - Closing of the semilunar valves
  - Opening of the A-V valves

Diastolic Sounds

Third sound Rapid passive filling of the ventricles

Fourth sound Rapid active filling of the ventricles due to atrial contraction

Since the cardiac chambers are filled with blood, none can vibrate without producing movements or vibrations in the blood which they contain Therefore, the cardiac walls, the valves, the arterial walls, and the blood represent an interdependent system which vibrates as a whole (Rushmer)

The mechanism producing the complex of the **first sound** has been repeatedly investigated, but the conclusions of the various researchers are by no means in agreement

Several authors suggested a purely muscular origin of the first sound Others, particularly Dock and Kountz and co workers, believe that the first sound is due to the sudden tension of the previously slack fibers of the A V valves A theory of mixed origin was advocated by Wiggers, who denied the possibility of separating in the tracing the vibrations caused by the various structures He postulated that vibrations were set up in the A-V valves, the chordae and the ventricular walls

The phonocardiographic studies of Orias and Braun Menendez and Rappaport and Sprague led to the view that the first sound is due to four separate factors (atrial, muscular, valvular and vascular) However, while two larger vibrations were recognized as coinciding with the two main valvular events, no separate vibration or group of vibrations was found to result from muscular contraction

Experimental graphic studies of Luisada, Alimurung and Lewis proved that (a) the two main vibrations of the *empty heart* are extremely faint and barely appreciable, and, (b) the first sound is the result of both muscular and valvular factors, a sudden change in muscular tension first closing the A-V valves and then opening the semilunar valves This causes a

double vibration of the cardiac wall, including high frequency and low frequency components, which are further transmitted to the chest wall Although simultaneous with the action of the valves, these vibrations are likely to arise in both the valvular and the muscular structures as a response to rapid changes in tension and pressure In other words, the first sound is the audible expression of that complex movement of the heart which is also revealed by the first part of the apical thrust

The mechanical events taking place at the beginning of systole were revealed in the sound tracings of the past by *two* large vibrations However, studies by Luisada et al (1957) in dogs and humans with a highly sensitive microphone and a cathode ray oscilloscope have revealed *four* vibrations Double catheterization of normal dogs under chloralose anesthesia have revealed the following facts

(a) There is a slight asynchronism in the rise of pressure between left and right ventricles the left is steeper and starts slightly earlier than the right The left ventricular rise (closure of the mitral valve) coincides with the *first* vibration of the first sound, the right ventricular rise (closure of the tricuspid valve) coincides with the *second* (Fig 4 A)

(b) There is a slight asynchronism in the rise of pressure between pulmonary artery and aorta the PA rise occurs earlier and coincides with the *third* vibration of the first sound, the AO rise occurs slightly later and coincides with the *fourth* vibration of the first sound (Fig 4 B) Therefore, the following events can be seen within the first sound complex

(1) Slow initial vibration = tension
(2) First rapid vibration = closure of mitral valve
(3) Second rapid vibration = closure of tricuspid valve
(4) Third rapid vibration = opening of pulmonic valve
(5) Fourth rapid vibration = opening of arotic valve
(6) Slow final vibrations = flow into the large arteries

The explanation of the slow initial vibration is based on the fact that it coincides with or follows the peak R of the ECG and slightly precedes the rise of pressure in the left ventricle The explanation of the final slow vibrations is based on their coincidence with the ascending branch of the aortic and pulmonic pulses

Smaller vibrations possibly of *mu cular nature*, may be seen in certain tracings between the two main groups of vibrations of the first sound but have usually a secondary importance *Vascular vibrations*, already admitted occur later after the beginning of ejection, and can be easily recognized because they are usually lower pitched

In conclusion, in spite of the existence of multiple factors and the fact that vibrations arise in multiple structures, the first heart sound has four clearly visible phases which coincide with the two main valvular events taking place in early systole

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In conclusion, in spite of the existence of multiple factors and the fact that vibrations arise in multiple structures, the first heart sound has four clearly visible phases which coincide with the two main valvular events taking *place in* early systole

The *second sound*, as *clinically heard, is caused by the closing of* the semilunar valves of the aorta and pulmonary artery However, subsequent vibrations coinciding with the opening of the A-V valves may be seen in graphic tracings *and may, in certain cases, either prolong the* sound or become audible as a separate sound

Experimental studies of Luisada et al (1957) have shown that the incisura of the *pulmonic* pulse takes place after that of the *aortic* pulse This indicates that, whenever two large vibrations are visible within the second sound, the first is aortic, the second pulmonic This confirms clinical observations of Leatham (1954)

The *opening of the A V valves*, according to the same study of Luisada et al (1957), is also nonsimultaneous Opening of the tricuspid valve precedes that of the mitral valve This explains occasional multiple *vibrations which can be observed in early diastole* between second sound and third sound

The third sound is a dull sound which may be heard at the apex of normal children or adolescents and either at the apex or epigastrium of cardiac patients

Two alternative theories have been advocated The first, advocated by Thayer (1909) and by Dock (1956) attributed this sound to a vibration of the mitral valve while other authors even attributed it to an impact of the apex on the chest wall A second theory explained it as due to vibrations of the ventricular walls at the time of rapid filling on account of the onrush of blood from the atria to the ventricles The perfect coincidence between third sound and peak of the phase of rapid filling seems to confirm the last theory Revival of the concept of *active diastole* of the ventricles might lead to speculation that the sound takes place at the maximum of this phase or at the time of its cessation Luisada (1952) observed splitting of the third sound in clinical cases Experiments in dogs reveal that rapid filling takes place much earlier in the right than in the left ventricle (Fig 3 B) In clinical cases, a third sound due to the left ventricle is usually heard or recorded at the apex while a third sound due to the right ventricle is heard or recorded at the epigastrium or at the midprecordium

The fourth sound takes place in presystole and is due to vibrations of the ventricular walls which occur when blood is forced into the ventricles by atrial contraction Comparison of the fourth sound recorded by way of the esophagus with that recorded at the apex revealed that while early vibrations of the esophageal tracing could be attributed to the atrial contraction *per se* or to valvular vibrations, the vibrations recorded at the apex occurred later and could be explained only with ventricular vibrations (Orias and Braun Menendez, 1939)

As right atrial contraction slightly precedes left atrial contraction, a double fourth sound may be recorded in clinical cases (Iwasada 1952)

THE ARTERIAL PULSE

The left ventricle propels its blood into the aorta at each beat This vessel stores a portion of the blood received so that neither the pressure nor the flow fall too low before the next ventricular contraction The aorta offers little resistance to the flow of blood Its great distensibility, however, gives a variable resistance according to the rate at which the pressure changes e g the heart rate

When the aortic pressure rises suddenly during the ejection phase of ventricular systole because of sudden addition of blood expelled from the left ventricle the aortic volume increases considerably, creating a new space (*aortic reservoir*) When the pressure falls during ventricular diastole the recoil of the wall can be compared to the reinjection of blood from the reservoir into the aorta so that the pressure tends to be maintained in spite of the lack of flow from the heart In addition to this change in the size of the aorta the *systolic discharge* of the left ventricle succeeds in causing a forward movement of blood The progressive expansion of the arterial wall from the center to the periphery reveals this movement

The *pressure wave* caused by the contraction of the heart *travels with* a speed of 3 to 4 meters per second in the aorta and 7 to 14 meters per second in the peripheral arteries This speed is much greater than the average rate at which the blood flows toward the *periphery (14 to 18* cm per second) The difference disappears in the capillaries, where the pulsating pressure is converted into steady pressure and flow

At the closure of the semilunar valves the recoil of the aorta maintains the onward drive of the blood At this time the peripheral arteries are still undergoing distention but they return to a smaller size as the excess of blood flows through the capillaries

FUNCTIONS OF THE VEINS

The return of blood through the venous system is only partly due to remaining force after it has passed through one or more capillary systems Many different mechanisms have been recognized which favor the venous return (1) contractions of the veins (2) decreasing pressure in the large veins due to the action of the heart (systolic suction) (3) aspirating effect of the low pressure existing in the thorax and (4) action of skeletal muscles on the nearest veins

CHAPTER TWO

Technique of Catheterization

THE PATIENT

Preliminary Studies

Whenever a patient is referred for cardiac catheterization, one or more members of the *catheterization team* should visit and examine the patient Routine blood and urine examinations, as well as electrocardiograms and phonocardiograms, should be ordered Fluoroscopy and roentgenogram of the chest in the *posterio anterior, right and left anterior obliques*, and in a lateral view* with barium swallow, should be done Electroky mograms or roentgenkymogram of the left atrium (or other regions) may be also selectively taken

A clinical diagnosis, based on the history, physical examination, and laboratory *tests, is then made This is necessary in order to decide* whether or not catheterization should be performed and which type should be preferred (right, left or both) If no clinical entity is recognized, the referring physician should be informed that the extensive preliminary tests do not justify the undertaking of catheterization The patient should later be informed that, although there is a murmur over the heart simulating that of an organic heart lesion, the laboratory tests suggest that no heart lesion is present

If the clinical diagnosis is that of a congenital lesion of the heart, the procedure should be explained in layman s terms to the patient, the parents or immediate members of the family The possible hazards of cardiac catheterization should not be underemphasized A good relationship between the patient (or parents) and the members of the *catheterization team is necessary in order to alleviate unnecessary* anxiety

If the clinical diagnosis is that of rheumatic heart disease with either aortic or mitral valve involvement, the procedure should be explained to the patient with a brief but accurate description of the test

The team. The procedure of catheterization is carried out by a team which should include as a minimum the following persons

(a) *The chief of the laboratory of* catheterization who prepares the vein (or the chest) introduces the catheter (or the needle) and manipulates the catheter under fluoroscopic control He also supervises the work of the team

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* This projection should be *utilized in the case of left heart catheterization*

(b) A physician (a resident or a research assistant) who uses the recording instruments and pays special attention to the electrocardiogram of the patient and to the details of the pressure tracings throughout the entire procedure

(c) An x ray resident (or a research fellow) who handles the x ray apparatus following the directions of the chief

(d) A chemistry technician who collects the blood samples and is responsible for the blood chemistry (gas analysis)

If other personnel are available another research fellow or resident may collaborate by handling the manometers and taking care of the photographic tracings

Whenever a *child* of 5 years or more is catheterized, a nurse can collaborate by being present at the procedure, taking care of the comfort of the little patient, talking to him and reassuring him In the case *of infants* or younger children, an anesthetist is required to attend (the entire procedure is undertaken while the patient is under basal anesthesia) An assistant may help to prepare one of the branches of the femoral vein or the great saphenous vein of the right side

The catheterization team may consist of from 4 to 8 persons

The usual precautions against excessive *radiation*, both to the patient and the observer are observed However the operator wears no lead gloves and should avoid exposure of the hands to x ray

Radiation hazard Two partitions made of lead plates can be placed at the sides of the patient's arm along the edge of the x ray table This device will reduce excessive secondary radiation from the body of the patient to the bare hands of the operator While trying to locate the point of introduction for the needle in an intercostal space on the back, in left heart catheterization, a 12 inch long metal clamp may be used to reduce excessive radiation A radio sensitive *film badge* for the detection of accumulated radiation should be worn by every member of the team Such a badge should be attached to the lower part of the right arm of the person who guides the catheter In order to avoid excessive radiation to the patient especially in infants (who may receive 80 per cent total body radiation if the fluoroscopic screen is widely open), the head and neck of the patient as well as the body below the diaphragm, should be protected by soft lead rubber plates

In our laboratory, the total duration of fluoroscopy in right heart catheterization is about 3 to 5 minutes and seldom exceeds 8 minutes with a 60 KV 2 MA for children and 70 to 80 KV, 2 5 to 3 MA for adults For left heart catheterization, the fluoroscopic duration varies from 1 to 3 minutes, and for combined heart catheterization it requires from 5 to 6 minutes

A fluoroscopic *image intensifier* has been used in a few medical centers The *advantages* of this device are (1) There is a reduction of total radiation to the patient and the team workers and (2) the procedure can be performed under ordinary dim light The *disadvantages* are (1) The size of the image is small, (2) the image is reversed, and (3) the device is

expensive This intensifier is certainly helpful in laboratories where more than 4 weekly procedures are being practiced and radiation hazard is still great, especially if angiocardiography is associated with catheterization

Preparation of the Patient

In order to obtain a basal metabolic state during the procedure and to avoid untoward regurgitation from the stomach and other complications, fasting for 4 to 10 hours is necessary both in adults and children, and no feeding is given to infants for 4 to 6 hours prior to the procedure

Procain G Penicillin is given for prophylactic reasons 600,000 units are injected intramuscularly 12 hours and 1 hour before the procedure, the same dose is also given 24 hours after the procedure In case of hypersensitivity to *Penicillin*, a broad spectrum antibiotic can be given

Sedation with Pentobarbital, Seconal or Nembutal (100 mg orally) is seldom necessary in *right* heart catheterization of adults In the *left* heart catheterization, moderate sedation and analgesia usually give better results This can be accomplished by giving 100 mg of *Pentobarbital* orally, one hour before the procedure, plus 50 to 100 mg of *Meperidine hydrochloride (Demerol)* intramuscularly, just prior to the procedure In children from 4 to 10 years of age, *Seconal* 60 mg orally, one to two hours before the procedure and *Meperidine hydrochloride* 20 to 30 mg, intramuscularly, one half hour before local anesthesia, are employed They usually give adequate sedation for about two hours In infants, basal anesthetics, such as *Pentobarbital** (rectal suppository) or *Avertin* are given If the child becomes restless, a small amount of *Demerol*, 10 to 20 mg, may be given intravenously

Intravenous infusion of 1 25 to 2 5 per cent *Thiopentone* or other short acting barbiturates can be used The use of intramuscular *Thiopentone*, even though found satisfactory by Fisher et al, may be complicated by damage to nerves or sloughing off of tissue

The patient lies *supine* in right heart catheterization and *prone* (or in a *left lateral position*) in left heart catheterization He is on a fluoroscopic table covered by a one inch foam rubber pad During the procedure the patient may be turned slightly to the left or right to obtain right anterior oblique or left anterior oblique views in *right* heart catheterization The patient may be turned from the prone to a lateral or the supine position in *left* heart catheterization after withdrawal of the needle while the catheter tip is left in the desired location within the left ventricle or atrium The patient should never be put in a head up position for more than a few minutes during or immediately after the procedure

* The dose is 4–5 mg /lb not exceeding 100 mg total

This is especially important in those patients who received sedation, in this position they are liable to develop transient hypotension loss of consciousness or a transient respiratory arrest Fortunately, these conditions are reversible by returning the patients to the supine position

When both right and left heart catheterizations are performed in the same subject, either can be performed first If right heart catheterization is performed first after recording the pulmonary artery wedge pressure, the catheter should be withdrawn into either the left or the right pulmonary artery The patient can then be turned to a prone position for left heart catheterization On the other hand both right and left heart catheterizations can be performed in a prone position by inserting two needles one into the left atrium one into the right This double puncture is not recommended by the authors because of additional hazards (see below)

THE CATHETERS

Right Heart Catheterization

Standard radio opaque cardiac catheters No 5 6 and 7, short (100 cm) and long (125 cm) should be prepared Larger lumen radio opaque cardiac catheters Nos 6 7 or 8 primarily designed for arteriography, can also be used These catheters are softer and may require a metal stylet to increase their rigidity for introduction into the right atrium They are more flexible and it is easier to make a half loop with them and to rotate them The larger lumen facilitates the collection of blood samples and the performance of selective angiocardiography in the various chambers Also there is less tendency to formation of a fibrin clot Regular No 5 catheters are used in infants while No 6 arterial catheters are prepared when the size of the saphenous vein is adequate Other catheters are used in adults according to body size and caliber of the vein A small curvature near the tip of the catheter permits the direction of its progress through rotation of the external end The latter is fitted with an adapter similar to the hub of a hypodermic needle

The catheter can be sterilized by autoclaving or by boiling for 30 minutes in distilled water After sterilization it is wrapped in a sterile towel It should be tested for surface imperfections or leaks before introduction After use it should be rinsed with tap water, filled with Haemosol solution and allowed to soak in the same solution for one hour Then it should be rinsed for one hour with tap water through a pressure connection This procedure serves to prevent deposition of particles of blood Sterilization of the catheter with antiseptics may cause febrile reactions and even pulmonary thrombosis and infarction (Dexter)

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cava By changing the position of the arm and rotating the catheter the tip can usually be advanced into the superior cava If the right basilic vein is used this difficulty is usually avoided On the contrary when using the cephalic vein the operator usually fails to manipulate the catheter into the thoracic inlet because of a tortuous angulated course before the subclavian vein is reached (Fig 6) One should not spend more than one or two minutes of fluoroscopy in attempts to advance the same catheter from the arm into the thorax Instead the operator should abandon the cephalic vein and try the basilic vein of either the same or the other arm Vision of the field by the patient is prevented by use of a small screen or a pillow

The skin is sterilized and infiltrated with 1 per cent *Novocain* then a sterile tourniquet is applied above the elbow A transverse incision about 1 cm in length is made (a longitudinal cut might obviate the need for suture but may be followed by unpleasant after effects and therefore is not used) The vein is isolated by blunt dissection and a double loop of silk is passed under it The silk is divided and one piece is pulled to the distal end of the exposed part and tied with a single knot to prevent retrograde bleeding

While the wound is covered with sterile gauze saturated with saline the vein is elevated and a small fish mouth incision is made with a small strabismus scissors The *catheter tip* (checked for slow constant rate of dripping and lubricated with normal saline solution) is then inserted into the vein often with the aid of a fine curved dressing forceps Next it is pushed several centimeters into the lumen of the vein Further progress of the catheter is carried out under fluoroscopic control If obstruction is met on attempting to enter the chest rotation of the catheter or slight movements of the arm and shoulder usually allow it to proceed The catheter is directed first into the superior cava then into the right atrium (Fig 6) If the tip enters the inferior cava it should be withdrawn to the right atrium and then slightly rotated and advanced

A vein of the lower limbs can also be used and is preferred whenever the veins of the arm are thin or thrombosed or the skin is edematous or inflamed In infants the veins at the elbow are so thin that a vein of the right inguinal region is always preferred The great saphenous vein a branch of the femoral vein or the main femoral vein is then prepared and used for catheterization (Fig 7)

In order to identify the great saphenous vein a point should be found *below* the inguinal

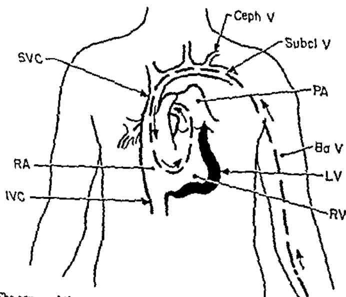


Fig 6—The course of the catheter for right heart catheterization through a vein of the elbow

Left Heart Catheterization

The outer diameter of the catheter should be such as to fit without difficulty in a No 18 thin walled needle There are three kinds of non radio opaque catheters polyethylene catheters size PE 50, No 442 T polyvinyl tubes and nylon catheters The length of the catheter for *retrograde arterial catheterization* to the ascending aorta is 22 inches (56 cm), for left heart catheterization it is 20 to 22 inches (51 to 56 cm) If a longer catheter is introduced into the left atrium without entering the left ventricle, an undesirable knot may be formed in the atrium Thoracotomy may then be necessary for its extraction Nylon and polyvinyl catheters are stiffer and may be successfully used in order to enter the left ventricle in pure, severe mitral insufficiency On the other hand, when using one of them, part of the catheter may be easily caught or cut off by the sharp end of the needle The authors recommend the use of a polyethylene catheter

THE NEEDLES

A regular 20 gauge 1½ inch long needle is used for collecting one sample of arterial blood and for a single pressure measurement in a peripheral artery A Cournand arterial needle is preferable for collection of several samples of arterial blood at long intervals and for multiple pressure measurements in a peripheral artery An 18 gauge, thin walled, 1½ to 2 inch long, needle is used for *retrograde arterial catheterization* with small polyethylene catheters An 18 gauge, thin walled, 6 inch long needle is used for *transthoracic puncture of the left atrium* In some instances, the use of a 7 inch long needle may be necessary to reach the left atrium (thick chest) A 17 gauge, thin walled needle has been used by others to obtain simultaneous pressure tracings from the left ventricle (through a polyethylene catheter) and from the left atrium (through the space between the small catheter and the needle wall) The authors hesitate to use such a large needle because of greater danger of hemorrhage Special care should be taken to blunt the inner sharp area of the needle with a sharpening stone pencil * This blunting will decrease the chance that part of the catheter may be either caught or cut off during withdrawal

THE SURGICAL PROCEDURE

Right Heart Catheterization

Any vein of suitable size which runs into the median basilic vein of either the right or the left arm can be used The left arm is preferable because the catheter follows a natural curve and the tip advances more easily from the right atrium into the right ventricle and out into the pulmonary artery In children it may be difficult at times to pass the catheter from the left subclavian into the left innominate vein and the superior

*HF 863 HARD Arkansas U S A

le ser upward turn in order to enter the right ventricle from the right atrium After entering the right ventricle unless the tip of the catheter is now turning headwards further advancement is likely to induce ventricular premature contractions and does not bring i into the pulmonary artery Therefore a small half loop (HL Fig 8) is made while the catheter tip is still in the right atrium However at times the catheter tip may hit the septal wall of the right ventricle if the angle of the half loop is too large or the crista supraventricularis if the angle is too small In each instance forced advancement should be avoided and on the contrary the catheter should be slightly withdrawn Slight rotation of the catheter usually allows the catheter tip to face the pulmonic orifice instead of the ventricular wall A gentle push at this moment permits it to enter the pulmonary artery The tip is then pushed into one of the main pulmonary stems and further on into one of the secondary pulmonary branches (Figs 6 and 7)

As *venous spasm* developed early in the procedure may prevent introduction or manipulation of the catheter it is advisable to advance the tip as far as possible into the *pulmonary artery or in a wedge position* Thus pre sure recordings and blood sample collections should be accompli hed *during withdrawal* In order to prevent venous spasm isolation of the vein should be gentle and lubrication of the surface of the catheter should be repeated from time to time especially before any further advancement of the catheter Too large a catheter in erted into a small vein or incomplete sedation especially in children may cause diffuse venous spasm

The following records can be taken (a) pulmonary artery wedge tracing (b) main pulmonary artery tracing (c) right ventricular tracing, (d) right atrial tracing and (e) superior or inferior caval tracings Of less common use are pressure measurements *in the coronary sinus and in the renal and hepatic veins* Also, one can record a *hepatic vein wedge pressure tracing* Pullback tracings from one location to another should always be taken from (a) to (b) (b) to (c) and (c) to (d)

When the catheter loop forms an acute angle within the lower part of

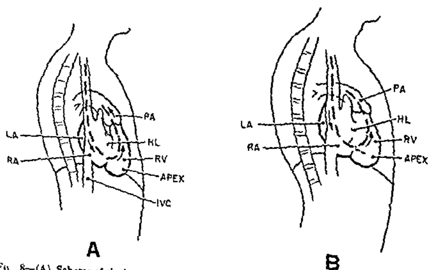


Fig 8—(A) Scheme of the heart in a lateral projection showing the course of the catheter in the right heart
(B) Scheme of the heart in a right oblique projection showing the course of the catheter

ligament and *medial* to the femoral artery according to the following description The *distance from the ligament and the artery* varies according to age

Age 1-6 months	1¼ fingers (1 5-2 cm)
Age 6-18 months	1½ fingers (2 5 cm)
Age 1½-5 years	2 fingers (3 0-3 5 cm)

A transverse incision 1½ cm long is then made across this point under local anesthesia The great saphenous vein is usually located deeply under a longitudinal line *passing through this point The catheter is passed from the femoral vein into the iliac* veins then into the inferior cava until it reaches the right atrium Once there a complete inversion of its course is necessary for entering the tricupsid valve Whenever the catheter tip is facing backwards further pushing would make a knot or a loop in the right atrium

In the supine position the catheter has to turn not only to the left (Fig 6) but also upwards (Fig 8 A) in order to enter the right ventricle from the right atrium One usually asks the patient to turn slightly on his left side thus obtaining a right anterior oblique position for fluoroscopy (Fig 8 B) In this position the obscured shadow of the spine is avoided and the exact position of the tip of the catheter (pointing forwards up or down) is accurately determined Also in this position the right atrium and the right ventricle lie almost on the same horizontal plane In other words the catheter makes a

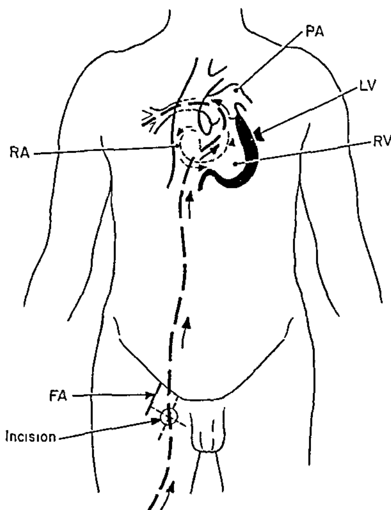


Fig 7—The course of the catheter for right heart catheterization through a vein of the lower limbs

Arterial puncture The syringe with the above described needle and 0 1 ml of heparin is held at an angle of 45 degrees in the right hand and the skin and subcutaneous tissue are entered at the site of *Novocaine* infiltration The needle is pushed forward at this angle toward the course of the artery which is identified by the middle and index fingers Arterial wall penetration is usually felt as a sudden decrease in resistance Bright red arterial blood should now enter the syringe with pulsatile flow Once the needle is in the lumen of the artery its position should not be altered until the desired quantity of blood is collected by gentle decompression of the plunger with the left hand In many instances the needle may penetrate both sides of the arterial wall In this case needle and syringe should be withdrawn slowly until bright red blood appears in the syringe If only one blood sample is necessary the needle is quickly withdrawn after sampling and a loose large gauze or sponge is quickly and forcefully applied over and above the site of needle entrance 2 to 3 cm above the skin puncture Heavy pressure over the artery should be maintained for at least 5 minutes If blood or a hematoma appear at the puncture site upon release of the pressure another 5 minutes of pressure are indicated If more time is necessary a firm bandage is applied over the gauze or sponge with the pressure point over or about the puncture site (never below) The needle is then disconnected from the syringe and the latter is made air tight by closing it with a metal cap filled with mercury *The blood* inside the syringe is mixed with the heparin solution (0 2 ml) by shaking the syringe for one to two minutes Any air bubbles appearing around the heparin solution should be removed before connection of the metal cap to the syringe Since the air bubbles usually contact the heparin solution only the arterial blood being collected will not be affected

In *multiple blood sample collection* a Cournand arterial needle is inserted in the same manner *except that it is usually* not connected to a syringe When bright red blood appears in the needle bore the smaller inside needle is removed and a blunt stylet is inserted into the lumen The angle of the needle should be changed to about 20 to 25 degrees before the needle with the stylet is advanced along the course of the arterial lumen The stylet is then removed to determine whether the needle is still in the artery as revealed by a pulsating flow of arterial blood Blood samples may be collected at varying intervals as described for a single blood sample

When an 18 gauge 1½ inch thin walled needle is used and a polyethylene catheter size I E 20 is to be introduced and left in arterial lumen (such as in left heart catheterization) the proximal end of the needle is held by middle and index finger and the stylet is pressed in place with the thumb It is most important to alter the angle of the needle to 20 to 25 degrees when arterial blood appears after temporary removal of the stylet The needle with stylet is then advanced approximately ¼ to ½ inch If the needle forms an angle of 45 degrees with the artery it is difficult to introduce the catheter and the patient usually complains of sharp pain A polyethylene catheter is then introduced into the bore of the needle and advanced to the desired position as soon as the stylet is removed

The position of the polyethylene catheter can be identified by injection of 1 ml of 70 per cent contrast medium (*Urokon* or *Neo iopax*) into the catheter The needle is then withdrawn and the catheter is left in place A large sponge or loose gauze is applied with pressure to the site of catheter entry into the artery Sustained pressure can be effectively maintained by adhesive tape or elastic bandage The catheter is now attached through a three way stopcock to the sampling syringe and a bottle of heparinized saline is placed at a level higher than the mean arterial pressure This will maintain a continuous *drip* between sampling periods Because of lower clotting tendency patency of the catheter can also be maintained by intermittent flushing every 10 or 15 minutes with heparinized saline On sampling the first ½ ml of blood should be discarded that following should be collected in the usual manner

Technique of collecting arterialized capillary blood The patient s hand is either wrapped in a hot towel or immersed in water at 45 C for 15 to 20 minutes The middle or fourth finger is dried with gauze and cleaned with 70 per cent ethyl alcohol A 4 mm deep finger puncture is made by a knife blade held at an angle of 30 degrees Arterialized capil

the cardiac shadow, *the coronary sinus* (Read et al) is frequently entered A blood sample taken from this point* and a pressure tracing taken after a slight withdrawal of the catheter usually allow the operator to recognize this location

It is not uncommon for the catheter tip to pass through *the foramen ovale* or *a small atrial septal defect* into the left atrium, especially if the catheter is introduced from the great saphenous vein in infants Usually, there is no significant evidence of left to right shunt between the two atria in such cases

In cases with *atrial septal defect*, the catheter can be passed from the right to the left atrium A left atrial tracing and a pulmonary vein wedge tracing may then be recorded The left ventricle may also be entered

In cases with *ventricular septal defect and overriding aorta*, an aortic tracing can be recorded by pushing the catheter from the right ventricle into the aorta

In cases with *patent ductus arteriosus*, the catheter occasionally can be passed from the pulmonary artery into the aorta

Arterial Puncture

Preparation of Syringe A 5 or 10 ml Luer look syringe is lubricated lightly and evenly with petroleum jelly (vaseline) before autoclaving Excess lubricant in the barrel or on the end of the plunger should be avoided because of possible air bubble entrapment which would prevent airless withdrawal of blood A 20 gauge 1½ inch needle is connected to the syringe and 1 ml of heparin (1000 unit per ml) is drawn into the barrel The syringe is then pointed upward the plunger is pulled to full capacity and then is returned to the 1 ml mark The maneuver wets the barrel with heparin and removes any air trapped in the space between the barrel and plunger By changing the position of the syringe and slightly withdrawing and pushing the plunger the remaining air bubbles can be expelled Only 0 1 or 0 2 ml of heparin is allowed to remain in the syringe while the excess is transferred to another syringe and the procedure is repeated

Selection of Arteries In adults and children beyond the age of five the brachial artery proximal to the antecubital fossa of either arm is preferable because it is relatively superficial palpable and adequate in size The femoral artery just above the inguinal ligament is used in small children and infants as well as in adults in whom brachial arterial puncture has failed

Identification of the course of the brachial artery Palpation of the brachial artery by the left index finger alone at or about the site of needle entry is not sufficient because a slight deviation of the needle from the course of the artery may lead to failure to enter First at approximately 3 to 4 cm proximal to the proposed needle entry point the pulsation of the artery is carefully palpated by the left index finger The left middle finger is now used to mark this upper point while the left index finger is moved downward slightly above the needle entry site

Local anesthesia The skin is cleansed with a solution of *Zephiran* in alcohol Infiltration of the skin with ½ to 1 per cent *Novocaine* is performed by means of a 24 gauge ½ inch needle and a 5 cc syringe The subcutaneous and deeper tissues are also infiltrated with 1 per cent *Novocaine* with the needle at an angle of 45 degrees toward the course of the artery This step should not be omitted especially when the needle remains in the artery for long periods of time

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* The blood sample of the coronary sinus is much darker than that of the right atrium and has a very low oxygen content

arterial catheterization, but also through direct puncture of the arch The needle penetrates from the suprasternal notch

For *left heart catheterization in animals*, the animal is lightly anesthetized with intravenous nembutal A lateral position is preferable in experimental animals because a supine position is difficult to maintain, and because it gives a better view for easy identification of the location of the catheter tip The hind legs are separated and tied to the table A large branch of one of the femoral arteries, or the artery itself, is isolated under local anesthesia A No 7 cardiac catheter is selected for larger dogs and a No 6 for smaller dogs A bottle with a pressure of 200 mm Hg and a slow dripping method is used The catheter is introduced into the isolated artery through an incision and advanced to the arch of the aorta A small loop should be made by pushing the catheter while the tip touches the aortic valve The loop will pass as a whole through the aortic valve into the left ventricle during the succeeding ventricular systole This maneuver decreases the possibility of damaging the aortic valve and reduces the frequency of ventricular premature beats Al o, a *loop* is easier to make in the aorta than in the left *ventricular cavity* As soon as the catheter enters the left ventricle no further attempt should be made to push it into the left atrium, unless the tip of the catheter is pointing backwards and upwards, then the attempt to enter the left atrium is made immediately after the end of ventricular systole

Bronchial puncture The patient is given mild sedation with atropine and morphine (or with *Demerol*) Local anesthesia of the pharynx and larynx is performed by painting the throat with *Amethocaine hydrochloride* and by instilling a few drops of this in the trachea A bronchoscope is passed down to the carina which is sprayed with the local anesthetic A 5 to 6 cm long needle having a bore of 3 mm is fused to a metal tube 50 cm. long having a 2 mm bore This is connected with the manometer by 40 cm of polyvinyl tubing having a bore of 3 mm The needle of the cannula connected with the manometer and filled with saline heparin solution is introduced into the bronchoscope and with the solution dripping is passed through the anteromedial wall of the left main bronchus at the carina The needle penetrates about 4 cm before entering the wall of the left atrium which is directly under the bronchus (Fig 9) As the procedure takes place upon a fluoroscopic table, fluoroscopic control of the position of the needle can be made before recording the tracing This procedure proved to be devoid of danger

Bronchial puncture of the pulmonary veins in the dog has been described by Henschel et al This procedure requires a special instrument The authors on the other hand, prefer retrograde arterial catheterization or transthoracic left atrial puncture as modified and applied in their laboratory

lary blood will flow spontaneously and rapidly from the puncture The blood is collected in a 2 ml lubricated and heparinized syringe by means of a small plastic funnel while an assistant withdraws the syringe plunger according to the rate of blood flow According to Hultgren, the oxygen content of the arterialized capillary blood is almost identical with that obtained by arterial puncture If anaerobically arterialized capillary blood is preferred, the finger should be immersed in liquid paraffin contained in the plastic funnel In newborn infants, a 4 mm deep puncture along the side of the heel is made in a previously warmed foot

If the ear is used, it is heated by radiant heat for 10 minutes at an air temperature of 45°C measured next to the ear lobe The skin is cleansed with alcohol and punctured The arterialized capillary blood is caught in heparin or oxalate in a specially constructed funnel from which samples may be drawn with a micropipette

Technique of collecting 'arterialized venous blood The hand is immersed in hot water at 45 C for 15 to 20 minutes Arterialized venous blood is collected in an airless way from any convenient vein on the back of the hand Arterialized capillary blood is preferable in small children

Left Heart Catheterization

Pressure tracings of the left atrium, left ventricle and aorta have been recorded by means of different techniques based upon puncture of an artery, the left atrium or the left ventricle *During cardiac surgery*, a needle can be introduced into the left atrium (Munnell and Lam, and several others), the left ventricle (Bjoerk) or the aorta (Radner) With closed chest, a needle can be introduced into the left atrium *through the left bronchus* (Allison and Linden, Facquet), *from the suprasternal fossa* (Radner), or from the *right posterior thoracic wall* (Bjoerk, Kent and Fisher) Then, a fine catheter can be passed into the needle and pushed following the direction of the blood stream until a pressure tracing reveals that its tip has reached the desired chamber of the left heart or the aorta

For *left heart catheterization*, the technique consists of isolation and puncture of the left brachial artery with a thin walled 18 gauge needle, fitted with a stylet A very thin polyethylene catheter is then introduced into the artery and gradually passed into the aortic arch * A small loop is made while the catheter tip rests against the aortic valve after which it can be easily pushed through the aortic valve into the left ventricle during ventricular systole The best position for entering the left ventricle is with the patient lying on his right side The procedure entails some dangers, including rupture of the aorta occlusion of a coronary artery, rupture of an aortic leaflet and episodes of ventricular tachycardia or flutter However, with the foregoing details of technique a gentle hand in pushing the catheter, and good training with experimental animals (see below), the hazards are greatly reduced Plain catheterization of the brachial artery or the aorta does not entail any serious dangers

A *pressure tracing of the aortic arch* can be obtained not only through

* In order to visualize the transparent polyvinyl catheter one can inject 1 cc of a 70 percent radio opaque material into it

sagittal plane The direction of the needle should be checked frequently The left atrium is usually entered at 10 to 12 cm from the skin and bright red blood flows from the needle when the stylet is removed A polyethylene catheter is then introduced into the desired location

Prior examination of the chest films (right lateral and P A views) is essential for an exact determination of the position of the left atrium A barium swallow may be helpful Measurements include the distance from the skin of the back to the posterior border of the vertebral body and from the skin to the most prominent point of the cardiac silhouette (posterior wall of the left atrium) This measurement gives a gross idea of the depth of the needle under the skin in regard to possible puncture of the intercostal vessels and to reaching the left atrium The *center of the left atrium* is estimated by the crossing of

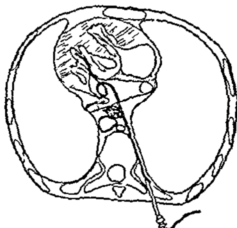


Fig 10—*Left* heart catheterization through the posterior chest wall (Courtesy of Bjork et al Am Heart J 1954)

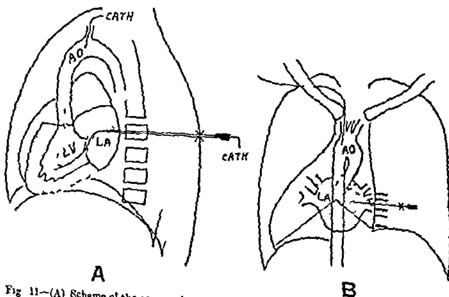


Fig 11—(A) Scheme of the course of the needle and catheter in left heart catheterization through the back (lateral projection)
(B) Projection of the left atrium on the posterior chest wall

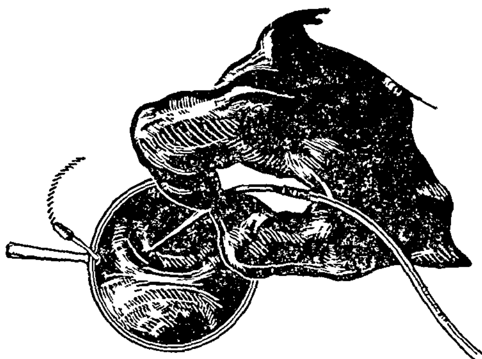


Fig 9—Transbronchial puncture of the left atrium Drawing of the view down the broncho scope showing the carina and the needle piercing the antromedial wall of the right main bronchus (Courtesy of Allison and Linden)

Chest Wall Puncture

The patient lies on *his left side* so that a right lateral fluoroscopic position is obtained After identification and localization of the left atrium a skin wheal is made with 1 per cent *Novocaine* over the ninth rib, about 5 cm to the right of the spinous process An 18 gauge, thin walled 6 inch long styletted needle is introduced along the upper border of the right ninth rib at the side of the vertebral body (Fig 10) The needle is then pulled back and reinserted along the vertebra until it enters the left atrium Bright red blood flows from the needle while the stylet is removed or can be aspirated with the syringe Fluoroscopic examination and a typical pressure tracing confirm the correct position of the needle A fine polyethylene (PE 50) or polyvinyl (No 442 T) catheter is then introduced through the needle into the left atrium It can be so manipulated that it enters first the left ventricle through the mitral valve then the ascending aorta through the aortic valve (Fig 11 A) Typical changes of pressure reveal the passing through each valve Upon completion of the procedure the needle should be removed first then the catheter in order to avoid the possibility of cutting the latter In the *right lateral* fluoroscopic position the authors found it difficult to identify the location of the center of the left atrium They also occasionally had difficulty in finding the intercostal space for insertion of the needle The authors found that *the prone position* advocated by Fisher is far preferable to a lateral In the former the intercostal space selected for the insertion of the needle varies from the seventh to the ninth intercostal space according to body build and cardiac size Puncture of the vertebral body may cause immediate discomfort and residual pain for several days and may be avoided through careful insertion of the needle

Puncture of the left atrium *in the dog* in the right lateral fluoroscopic view has been successfully carried out in our laboratory An intercostal space corresponding hori zontally to a point 3 to 4 cm above the lowest posterior cardiac border about 3 5 cm to the right of the spinous process is then used An 18 gauge thin walled 6 inch long stylet ted needle is inserted into this point making an angle of about 10 degrees with the median

saline solution Systolic vibrations are usually felt in cases with pure or predominant insufficiency while a diastolic thrill is seldom felt A polyethylene catheter is then introduced into the needle bore and advanced into the left atrium Heparinized saline solution is again splashed between the catheter and the bore in order to avoid the possibility of a blood clot around the entrance of the catheter The soft catheter is then advanced from the left atrium into the left ventricle At times the left ventricle is entered during a pull back of the coiled catheter in the left atrium therefore the operator should continuously watch the pressure tracing during manipulations If one fails to enter the ventricle in a few attempts the natural direction of the catheter should be slightly altered or the needle should be pushed one or two cm deeper into the atrium (if this is large on roentgenologic examination) These alternative maneuvers usually give a satisfactory result

In patients with predominant mitral insufficiency the left ventricle may be entered for only a few seconds as noted from the oscilloscopic monitor When the catheter is automatically and repeatedly thrown back from the left ventricle into the left atrium it indicates a *significant mitral insufficiency* A stiffer polyvinyl or nylon catheter usually succeeds to pass through the mitral orifice even when the polyethylene catheter had failed A further advance of the polyethylene catheter from the ventricle to the aorta is attempted only in patients suspected of having aortic valvular disease because *premature ventricular contractions* or short runs of *ventricular tachycardia* frequently occur during these attempts One should never employ stiff catheters for aortic *catheterization from the left ventricle* because of the severe risk involved Sometimes when the catheter makes a loop in the left atrium it suddenly enters the left ventricle for one beat then spontaneously advances into the aorta (Fig 12) This can be explained by the fact that the catheter making a half loop passed the mitral orifice with the tip facing the aortic valve During the subsequent ventricular contraction it flowed across the aortic valve

Hazards of Cardiac Catheterization

We have had no fatalities in either right or left heart catheterization However both procedures are by no means innocuous Serious and sometimes fatal complications have been reported in the literature (Goodwin, Nightingale Stern Cournand) The risk of fatal complications is about 1 per 1 000 in right heart catheterization and 5 per 1 000 in angiocardiography angiopneumography or thoracic aortography combined with catheterization which require general anesthesia Catheterization of the arch of the aorta usually causes no serious complications but one fatal outcome has been reported (Bagger et al) The risk of fatal complications due to left heart catheterization seems about twice to three times as high as that of right heart catheterization (this, however, includes the early stage of experimentation ' which preceded standardization of the method)

The causes of fatal complications in right heart catheterization, besides anesthetic death in infants and children, are *cardiac standstill, ventricular fibrillation bacterial endo myocarditis rupture of the ventricular wall air embolism pulmonary embolism rupture of a pulmonary arteriovenous aneurysm or of the coronary sinus* and *heart failure* In right heart catheterization and selective angiocardiography, one should *also* include *cerebral, vertebral renal* and *myocardial damage*

Although the causes of fatal complications in transthoracic left heart catheterization have not been adequately studied *hemopericardium*

two lines in the postero anterior view One line bisects the angle of the apex and is prolonged toward the right the other bisects the angle made by the right atrium with the diaphragm and is prolonged toward the left (Fig 11 B) Since the teleroentgenogram of the chest is usually taken in deep inspiration while catheterization is performed with normal respiration a slight difference may be noted After identification of the center or the left atrium, the corresponding intercostal space which is usually 1½ spaces above the highest point reached by the shadow of the right diaphragm is visualized and is marked by a long hemostat about 4 to 4 5 cm to the right of the spinous process A small wheal is made in this point The needle with stylet is inserted into the skin After removal of the stylet the needle is connected to a Luer lock vaselinized syringe containing 4 to 5 cc of 1 percent *Novocaine* The vaseline in the syringe does not allow the plunger to move spontaneously so that inadvertent injection of *Novocaine* into undesirable points *is avoided The needle and syringe are directed along an oblique line making an angle of* 20 to 28 degrees with the median sagittal plane The needle is pointing medially and should reach the left atrium at the center of its fluoroscopic projection During the insertion of *the needle, small amounts of Novocaine (0 2 ml every cm of penetration) are injected* after attempted aspirations Since the first part of insertion is performed without the aid of fluoroscopy the direction of the needle and stylet should be checked before entering the pleural cavity (about 6 to 8 cm from the skin) If the needle is pointing headward or too high puncture of the root of the ascending aorta might occur with further advance

It is the authors opinion that a through and through *puncture of an intercostal artery* may cause minor intrapleural hemorrhages Therefore the needle should be withdrawn and reinserted a few millimeters below the previous site If the direction is correct the needle syringe unit is then continuously advanced into the paravertebral and the posterior retrosternal spaces Occasionally a few tiny air bubbles may be aspirated due to *puncture of part of the lung tissue* A feeling of resistance is felt at the level of the posterior atrial wall (measured from the skin to the barium filled curvature of the esophagus) At this moment the patient may experience a feeling of dull pain Two further quick advances of the needle (5 mm at a time) usually allow it to enter the left atrium If it does not enter this chamber the speed of the last attempt of penetration may be too slow, therefore one should withdraw the needle by about 1 cm and try again However one should make certain that the needle is still in contact with the atrial wall This maneuver will decrease the undue invagination of the atrial wall and thus will avoid the possibility of *puncture of the right atrium* through the left atrium and the septum In one *case right heart catheterization was performed accidentally before left atrial catheterization* Rotation of the needle against the atrial wall is used to penetrate the latter without excessive invagination but it may damage the atrial wall and cause some pericardial hemorrhage

Bright red blood is aspirated and the needle may be unlocked after a safety stopper is tightened to the needle just above the *skin* of the back If the mean left atrial pressure is much higher than that of the hydrostatic pressure of the needle (about 12 mm Hg) bright red blood flows freely from the needle

If mean left atrial pressure is normal bright red blood will soon disappear from the needle bore because normal left atrial pressure is lower than the hydrostatic pressure of the needle It is not justified to advance further the needle without obvious reason because it may puncture the atrial septum the anterior mitral leaflet or the root of the aorta (when the left atrium is not too large) Accidental penetration of air into the left *atrium is possible if the patient takes an unexpected deep inspiration while the stylet is* removed (Bjork) It might cause *air embolism* into the systemic circulation This can be avoided by dripping heparinized saline into the space between the stylet and the needle bore before the stylet is completely withdrawn If the saline solution lowers its level rapidly the stylet is immediately reinserted Usually there is ample time for the operator to introduce the catheter into the needle bore before the saline level becomes invisible or to connect the pressure gauge to the needle bore

Direct pressure measurement of the left atrium can be made by connecting the strain gauge to the needle bore after withdrawing blood and flushing the needle with heparinized

with cardiac tamponade, due to puncture of the aortic root, *sudden death* and *heart failure* have been reported Other possible causes are *various cardiac arrythmias, massive pneumothorax, cerebral embolism, mediastinitis* and *bacteremia* No fatal complication was encountered with the transbronchial approach by Morrow et al

Major transient complications in right heart catheterization include convertible *atrial tachycardia, flutter* and *fibrillation, ventricular tachycardia* and *fibrillation, right bundle branch block, syncope, heart failure, intracardiac or vascular knot, endomyocardial damage severe pyretic reaction curable bacteremia* severe *thrombophlebitis* and *pulmonary infarction*

In left heart catheterization (Bagger et al Morrow), *hemopericardium,* moderate *pneumothorax hemoptysis,* amputation or knot of the catheter, short runs of *ventricular tachycardia* and *hemothorax* represent possible, non fatal complications

Many of the fatal complications may be reduced or prevented by careful selection of patients good technique and early recognition of serious complications

The general clinical condition of the patient should not be overlooked *There is no emergency in cardiac catheterization* It is safer to wait and treat patients who are in severe heart failure or poor general condition than to proceed Two such patients were presented to us The procedure was postponed because of the above objections The patients died within 36 hours from this decision!

Aneurysmal dilation of the aorta should be considered as a contraindication to transthoracic puncture of the left atrium On the other hand, valuable data may be obtained in such cases by resorting to *retrograde catheterization of the heart* Likewise, *myocarditis* or *acute pulmonary disease* usually represents a definite contraindication to right heart catheterization If valuable data may be obtained from this procedure, the judgment will depend upon the general condition of the patient and not upon the clinical diagnosis

It is universally accepted that the most important guarantee against fatal and serious complications is not only an experienced well organized and well equipped team but also a deep sense of respect for the patient Continuous observation of the patient s electrocardiogram and pressure pulses through oscilloscopic monitoring (or similar devices) and their recording (especially during each advance into or pullback from the ventricle) can immediately detect an otherwise serious arrhythmia The operator who manipulates the catheter should know not only the geographical anatomy of the heart but also the possible anomalies of the individual case In right heart catheterization the operator should identify the tip of the catheter before any further advance (see above)

Physicians of the team must be alert and possess good experience

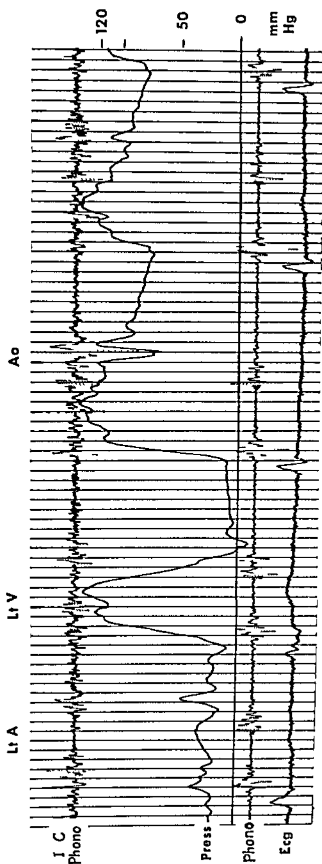


Fig 12—Advancement of the catheter in left heart catheterization (from left atrium to left ventricle then to aorta)
From above = intracardiac phonocardiogram
pressure tracing
external phonocardiogram
electrocardiogram

some of the commercial inductance transducers with little drifting of the baseline have become available Since some degree of drifting of the baseline as well as change in calibration factor, may occur even in the best available recording system, *the authors record the baseline before and after each pressure tracing* The calibration factor should be evaluated immediately after each pressure recording if the stability of the baseline and the calibration factor are poor On the other hand, in a stable pressure system, calibration before during and after each cardiac catheterization may be satisfactory

The accuracy of the pressure transducers for static and dynamic pressures are of great importance It has been shown (Fry et al) that the accuracy of the commonly used gauges to static pressure is usually satisfactory with linear response from 0 to 300 mm Hg However, the accuracy of the gauges to dynamic pressure is influenced by several factors Among them are the characteristics of the gauge the type of amplifier and the size and length of the needle and the catheter It has been stated (Hansen) that a satisfactory pressure tracing may be obtained by using a system which has from 70 to 80 per cent of critical damping

Critical damping of an electrical pressure recording system is defined as the amount of damping (friction or resistance) which is just sufficient to prevent oscillations like those occurring in a diaphragm displacement manometer *Overdamping* is any amount of damping which is *greater* than critical damping The pressure tracing is unusually smooth and the upstroke of ventricular or arterial systole is delayed It may not reach the actual systolic pressure level at the end of systole In other words, an overdamped ventricular systolic pressure is lower than its actual level Likewise the ventricular diastolic pressure is slightly higher than the actual diastolic level Even when the pressure transducer and amplifier have been used in a previous satisfactory procedure overdamping may still occur if the lumen of the needle or catheter is reduced to a critical level or the length of the needle or catheter is greater For this reason, a pressure transducer may be good for right heart catheterization but not for left heart catheterization where the inside diameter of the polyethylene catheter is much smaller than the No 6 or No 7 standard cardiac catheter A more sensitive strain gauge with higher natural frequency should then be used

Underdamping (overshooting) is any amount of damping which is *smaller* than critical damping Thus, oscillations appear in the pressure tracing and a slight degree of overshooting results As stated above the recommended damping in a pressure recording system is 70 to 80 per cent of critical damping At this level, slight overshooting of about 4 per cent may be expected for a short duration Likewise, the early diastolic pres

in all diagnostic and therapeutic measures against possible complications All members, including the technical personnel, should be trained to meet emergency situations Necessary drugs, such as a rapidly acting digitalis preparation, procain, amide, quinidine, caffeine, levo arterenol, epinephrine, *Isuprel* and oxygen, and necessary instruments for opening the chest in case of cardiac standstill, should be available An electric pacemaker would be desirable

THE APPARATUS

Intracardiac and vascular pressures can be recorded by several types of manometers including *water* and *mercury manometers*, as well as *diaphragm displacement type manometers* However, the need for easy recording of accurate pressure tracings from various chambers and at different degrees of amplification, and for higher frequency of response, led to the development of *electric pressure transducers* (Noble, Hansen)

The system consists of a recorder, an amplifier and a pressure transducer which is connected with a needle or a cardiac catheter

An electrical pressure transducer is a displacement gauge which transforms mechanical displacements into electric pulsations Several types of electric pressure transducers are commonly available, i e capacitance, inductance and resistance transducers and differential transformers *Capacitance* and *inductance transducers* usually give greater electric output or voltage than the *resistance transducers* or *strain gauges* Therefore, the reductance and capacitance transducers are more sensitive The electric output of the strain gauges is usually smaller because they are based on the principle that the electric resistance of the metalic film or wire conductor is altered by the application of pressure Due to the advanced development of resistance transducers, highly sensitive *strain gauges, suitable for left heart catheterization are currently available*

It is known that an *amplifier* for pressure transducers is necessary in order to obtain the various sensitivities which are desirable After amplification of the electric output of a pressure transducer the baseline may become unstable due to change in temperature of the gauge This will cause drifting of the baseline from time to time and also a change of the static calibration factor These changes would influence the accuracy of pressure measurements of a diagnostic catheterization Considerable drifting of the baseline occurs when direct coupled amplifiers for strain gauge output are used On the other hand such a drifting of the baseline becomes negligible when amplitude modulated carrier amplifiers for strain gauge are used For capacitance and reductance transducers, *phased* or *frequency modulated amplifiers* are used They often give some drifting of the baseline and change of the calibration factor However,

some of the commercial inductance transducers with little drifting of the baseline have become available Since some degree of drifting of the baseline, as well as change in calibration factor, may occur even in the best available recording system, *the authors record the baseline before and after each pressure tracing* The calibration factor should be evaluated immediately after each pressure recording if the stability of the baseline and the calibration factor are poor On the other hand, in a stable pressure system calibration before, during and after each cardiac catheterization may be satisfactory

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sure is slightly lower than its actual level However, insufficient damping is undesirable because, in the ventricular or arterial pressure tracings, *overshooting may be from 10 to 20 per cent of the actual systolic level* For this reason, a cardiac catheter of large lumen is undesirable for pressure recording systems if undue underdamping occurs A more rigid and sensitive pressure transducer is good for small lumen polyethylene catheters (as in left heart catheterization), but the pressure tracing becomes unduly underdamped when larger lumen catheters are used (as for right heart catheterization) However, by adjusting the three way stop cock in order to obtain a proper small opening or by inserting a 20 gauge needle of variable length between the larger lumen catheter and the pressure transducer, a desirable, slight underdamping pressure tracing may be recorded (Fig 13 A) The maneuver may reduce the frequency response of the system *Electric damping can also be used by the use of* a special "filter" (Fig 13 B) A "damping" needle parallel to the manometer and run out into free space was advocated by Vandertweel who claimed that the higher frequencies were preserved Since insertion of various size lumen needles is tedious, the procedure is not recommended by the authors

The Recorders

The tracings of pressure pulses can be recorded by either photographic or direct writing apparatus The authors have been using both types of recorders and found that a *cathode ray oscillograph with a DC amplifier and a photographic transcription* is the most sensitive, accurate and flexible recording device The flat frequency response of a cathode ray oscillograph usually runs from zero to several thousand cycles per second The *electric calibration* of such a channel is very convenient but should be periodically checked with a mercury manometer Mean pressure of arteries, atria or other chambers can be easily obtained by an electrical integration circuit A six channel cathode ray oscillograph* has been used in our laboratory It records accurate pressure tracings together with other simultaneous tracings used for timing purposes, such as the phonocardiogram and the electrocardiogram It also records other tracings which are of interest in studying the case such as the intracardiac phonocardiogram, the intracardiac electrocardiogram, the dye dilution curve or the oximetry curve This recorder has two multirace ' monitoring oscilloscopes The pressure pulse can be selectively placed on an oscilloscope for inspection and preliminary study at different speeds of 25, 50 or 100 mm per second The amplification can be in the range of 6 cm of vertical deflection for pressures of 10 20, 40, 80 160 or 320 mm Hg or any fraction thereof All interesting waves are shown on the other oscillo

* Built by Electronics for Medicine White Plains N Y

scope while the operator takes a tracing by a built in cathode ray moving film camera A defective tracing can be observed and avoided, thus rendering unnecessary a repeat of diagnostic or experimental catheterization

A *two channel electronic photographic recorder** with a DC amplifier and an *electromanometer*, connected to a separate oscilloscope, also gives accurate and flexible pressure pulses In this system, the baseline may drift and calibration factors may change due to changes of temperature The greatest disadvantage is that it allows to record only one other simultaneous tracing in addition to that of pressure

A *multi channel electronic direct writer*† with a DC amplifier and one or more electromanometers or strain gauges can also be used This apparatus is currently employed in our laboratory for animal experiments of long duration requiring extremely slow tracings (0 1 to 10 mm / sec) On the other hand it is definitely inferior to others because the maximal speed of the film is only 50 mm /sec and because of various inaccuracies which are inherent in the direct writing systems The direct arm recorder has a frequency response of not more than 80 cycles per second and is not adequate for recording heart sounds A direct writing recorder with higher frequency response (suitable for the study of heart sounds)‡ is also available but the reproduction of the records is difficult

A photographically recording galvanometer is the most simple and perfect type The most sensitive system consisting of a galvanometer coupled to a resistance pressure transducer requires no amplifier or a low gain carrier type of electronic amplifier The flat frequency response of these sensitive galvanometers is usually from 0 to over 100 cycles per second The *disadvantage* of this system is an unavoidable delay between the exposure of the film and the observation of the record If the record is defective it is often necessary to repeat the procedure However, *the repetition* is unnecessary if a cathode ray oscillograph with photographic recording or a direct writing apparatus are used One of the common objections to photographic recorders is the photographic processing On the other hand the excellent recording of heart sounds pressure pulses *etc* and the simple technique of film processing render this apparatus definitely superior to the others

RECORDING PRESSURE TRACINGS

The Conventional Method and Its Disadvantages

The conventional method is based on simultaneous recording of an electrocardiogram and a pressure tracing at a film speed of 25 mm /sec

* Sanborn Co Waltham Mass
† Poly viso Sanborn Co Waltham Mass
‡ Schwarzer Germany

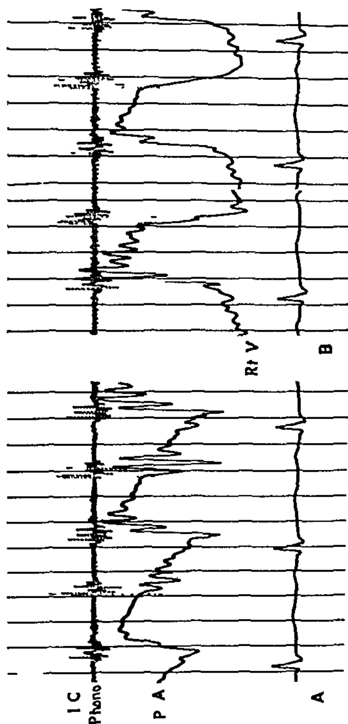


Fig 13—Damping of a high frequency strain gauge tracing
(A) Pulmonary artery—mechanical damping in the first beat
(B) Right ventricle—electrical damping in the second beat

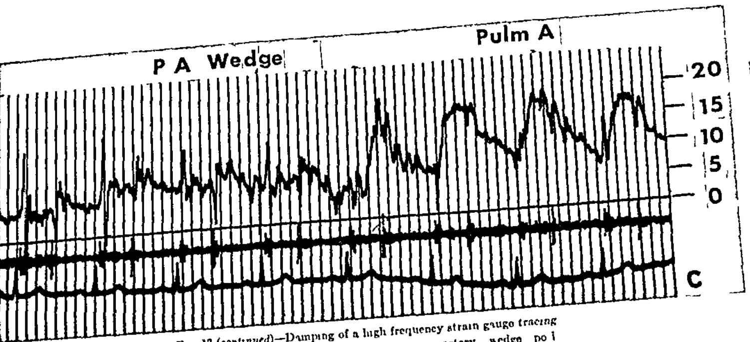


Fig 13 (*continued*)—Damping of a high frequency strain gauge tracing
(C) Pullback maneuver from pulmonary artery wedge position to main trunk of pulmonary artery

The amplification allowed is usually limited, resulting in waves between 5 and 15 mm high The tracing is usually taken on a direct-writing apparatus

Several objections can be raised against this type of recording

(a) *A wise balance between ordinate and abscissal values* is necessary for accurate observation of the details of the curve and of the levels of pressure Actually, this film speed is too rapid for a quick grasp of the pressure level, it is too slow *for a study of the patterns*

(b) A direct writer has several disadvantages including possible distortion of the tracing (modifications due to incorrect temperature, improper balancing, improper damping, or improper pressure of the stylus) and great vertical distance between the pressure tracing and that of other cardiac tracings used for "timing" the waves

(c) The electrocardiogram, though useful for supervision of the patient, is *inadequate as a timer* on account of a different "lag" between electric and mechanical phenomena,* possible lack of definition of the end of the T wave, lack of diastolic waves (except P), and possible *distorted complexes (bundle branch block, premature contractions, etc)*

Therefore, our experience led us to use a somewhat different technique which is described below

Suggested Method of Recording

Film speed Three speeds should be used according to the aim of the research Whenever one is interested in the *level of pressure* and in the *identification of the chamber* in which the catheter is placed, a slow film speed should be used † Speeds of 10 mm /sec or 25 mm /sec are usually the best They reveal clearly the typical changes in pressure during pullback maneuvers

(a) For right heart catheterization pull back from PA to RV and from RV to RA
(b) For left heart catheterization pull back from AO to LV and from LV to LA
(c) For left heart catheterization through brachial artery pull back from LA to LV and from LV to AO

Direct writers are adequate for this limited purpose

On the other hand, if one is interested in *the study of the patterns of the pressure pulses* photographic recording and more rapid film speed are necessary The photographic recording insures greater fidelity and amplification in the reproduction of the details High speeds (50, 75 or 100 mm /sec) cause a spreading of the waves with better observation and timing of the various accidents of the tracing Moreover, since the various simultaneous photographic tracings are not widely separated, like those

* A common error occurs in mitral stenosis in which the first sound is delayed over the QRS complex as first proven by Cossio

† A slow film speed has been employed by Whitaker and by Gibson and Wood

of the direct writers, and since they can even cross each other, timing of the waves is easy and accurate

It should be kept in mind that the slope of any arterial pulse wave is more or less steep according to the ratio of abscissa (increased by greater amplification) to ordinate (increased by greater film speed) Therefore, the best way to decide upon the rapidity of expansion of the pulse is to measure the ordinate distance between foot and peak and to relate that to the duration of systole Moreover, it is important to note the distance from the peak of the wave to the second sound

Simultaneous tracings for timing We have found that the best single tracing for timing purposes is the *phonocardiogram* The exact phase of the mechanical events of the heart, and chiefly the phases of ventricular systole and diastole can be easily identified if one uses as reference the heart sounds simultaneously recorded by means of a phonocardiogram Even the short phase of ventricular tension can be recognized in this way The use of the phonocardiogram will avoid certain common mistakes like that of attributing a slightly delayed A wave of an atrial pressure tracing to ventricular systole or confusing abnormal presystolic phenomena with those which take place during the extremely brief tension period of ventricular systole

If more than two tracings can be recorded the third should be an *electrocardiogram* The phonocardiogram is recorded by placing a closed chest piece at or slightly outside the apex This piece is connected by a short length of hard tubing to a microphone placed under the pillow The cable of the microphone is plugged into the recording apparatus and the tracing is recorded at high speed using the 'stethoscopic' method (good amplitude of the low pitched sounds)

More recently an extremely small (3 cm x 1 5 cm) flat crystal microphone was used in our laboratory *

Zero Reference Line or Baseline

An accurate recording of the manometric *zero reference* line or *base line* is necessary either before or after each recording of pressure tracing It is important to employ a satisfactory *standard level of zero reference* with which the operator becomes acquainted in order to permit comparison of values especially when both sides of cardiac catheterization are performed in the same subject but in different positions As the patients are usually in the supine or prone position during the procedure, *a point which is half way between the outermost anterior surface of the sternum at the third interspace and the outermost posterior surface of the chest is used* It practically corresponds to the level at which both venae cavae enter the right atrium and to the antero posterior central point of the

* Supplied by Electronics in Medicine of White Plains N Y

left atrium *A line passing through the center of the sternum and the center of the spine should be used when the patient lies on either the right lateral or the left lateral position for left heart catheterization*, because the antero posterior mid chest line is at about one half of the thoracic lateral diameter The authors have studied the left atrial pressure by using *the mid chest level* as zero reference line compared with *the tip of the needle* in the left atrium as zero reference The difference between the two varied from 2 to 4 mm Hg It is improper and difficult to compare the right and left atrial pressures by using the tips of the needles as zero references because they are at different depths from the skin of the back In other words, there are two different levels of zero reference, i e , one for the left atrium and ventricle, another for the right atrium and ventricle In cases of atrial septal defect, the use of the antero posterior mid chest line as a single zero line permits calculating the difference of pressure between the two atria without difficulty (Fig 14)

Roy, Gadboys and Dow have confirmed our experience that the midchest level is the most accurate zero reference level For the zero level in semi sitting or sitting positions of the patient, the "phlebostatic axis" and the "phlebostatic level" described by Burch are employed Pressure measurements by using all other reference baselines are un suitable for universal comparison unless the thickness of the chest wall is also given

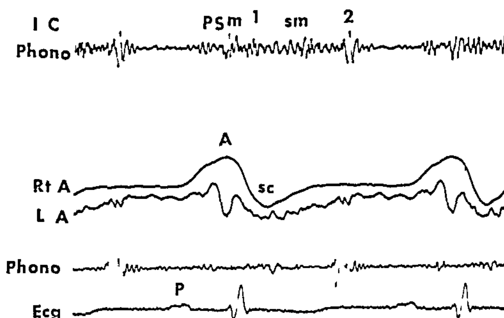


Fig 14—Simultaneous pressure tracings of the two atria obtained with two needles in a case with intact atrial septum (same zero line same calibration)

The highest tracing is an intracardiac phonocardiogram recorded in the left atrium Below external phonocardiogram and electrocardiogram A presystolic murmur (I S_m) and a systolic murmur (S_m) were recorded in the atrium

INTRACARDIAC ELECTROCARDIOGRAPHY

Unipolar Tracings

Intracavitary currents are usually obtained by introducing an exploring electrode within one of the cardiac chambers As exploring electrode, various devices have been used

(a) *A thin metal wire* is introduced into a cardiac catheter until it approaches the tip The blood interposed between the tip of the wire and the cavity acts as a conductor so that the results are practically the same as if the wire were within the chamber

(b) *Physiologic salt solution* contained within the catheter acts as a conductor and is used for the exploring electrode The procedure was first described by Hecht et al and by Hellerstein et al The authors have revived this method according to the following technique since the catheter is made of insulating material electric currents can be easily conducted from its tip to its end For better conduction the catheter and the pressure transducer are filled by means of a syringe *with sterile 5 per cent (hypertonic) salt solution with 10 per cent Heparin* (for the right heart 1 cc is necessary for the left heart 0 5 cc is sufficient) The wire of the chest lead is then connected with the stopcock of the strain gauge which acts as an exploring electrode Wilson's central terminal is used for closing the circuit The x ray and any electric appliance (except for the Kymograph) should be disconnected and the operating field should be as dry as possible in order to avoid AC interference

Bipolar Tracings

A different method was used by Luisada et al (193) A specially modified catheter ended in a silver tip a small silver ring was at 1 cm from the tip Both this tip and the ring were connected by means of insulated wires running inside the catheter with two binding posts at the proximal end The two posts were then connected to the ECG wires as in lead I Interesting tracings were recorded with this device

It should be kept in mind that the movements of the tip of the catheter may alter the tracing and that contact of the tip with the endocardium may cause an injury pattern even though a conventional lead may not show the latter (Fig 3?) Slight withdrawal of the catheter is followed by disappearance of such pattern

INTRACARDIAC PHONOCARDIOGRAPHY

Intracardiac phonocardiography (i c phono) is a recently described technique *Right* heart i c phono was independently described by Yamakawa et al Soulié Wallace et al and Moscovitz et al *Left* heart i c phono was described by Luisada and Liu by using a different principle which can be applied to either side of the heart

Yamakawa et al used a condenser microphone placed at one end of a catheter while the blood and body served as the second electrode This resulted in emphasis of the swishing sounds caused by the blood around the tip of the catheter To a lesser extent all intracardiac microphones have this same disadvantage

Wallace et al used a miniature tubular hydrophone of the type employed in undersea warfare (Sonar) A dual type catheter is used so that blood samples may be withdrawn during catheterization The response of the microphone seems to be between 180 and 20 000 cycles per second at a level of −140 db referred to one volt for one micro bar The response continues below 180 but is less accurate

The method described by the authors is as follows The catheter placed in the cardiac chambers or large vessels contains a column of fluid which connects the intrathoracic

organ with a transducer of high frequency response (Statham P 23 D) As water is a good conductor of sound waves (at a speed of about 1500 meters per second), it is possible to pick up vibrations of sonic frequency *at the proximal end* of the catheter It would be possible to record vibrations by a special type of hydrophone in this location However the sensitivity and the frequency response of the pressure transducer are such that the latter may be used for simultaneously recording both the pressure pulses and the sonic vibrations

The vibrations of sonic frequency are obtained through a triple electric differentiation and with the use of two variable band pass filters * A single section of the RC type provides differentiation for signals with frequency below 100 cycles which are fed to a lin ear" amplifier Fed to an amplifier with stethoscopic" characteristics, the signals receive a second differentiation Further fed to an amplifier with special filters, they receive a third differentiation Signals having frequencies higher than 100/sec pass undifferentiated but amplified The output of the 'differentiators' increases linearly with increasing frequencies up to 100 cycles/sec then gradually flattens off to give constant output The final output is obtained through the use of a low pass and a high pass filter Both cause the signals to decrease at a rate of 12 decibels per octave at frequencies respectively below and above those selected by the switches Bands of 60 to 110 60 to 250 or 60 to 500 were alternatively used

* The circuit for this setup was specially built by 'Electronics for Medicine' of White Plains, N Y

CHAPTER THREE

Normal Patterns of Pressure in the Cardiac Chambers and Large Vessels

The normal levels and patterns of pressure in the right and left heart are presented in Fig 15

Considering that the patterns of the two venae cavae largely reflect the waves of the right atrium (with some degree of distortion), the description should start from the latter

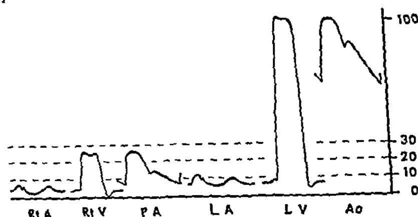


Fig 15—Scheme of normal pressures in the cardiovascular system

RIGHT ATRIUM

The right atrial pattern is basically formed by two positive waves, one in presystole (preceding ventricular systole) and the other during early ventricular diastole (Fig 16)

Presystolic Wave

The presystolic wave to be called A wave, is caused by the contraction of the right atrium It starts about 0 14" to 0 15" before the beginning of the first sound in adolescents and adults and between 0 12" and 0 14" in children Its peak always precedes the onset of the first sound The peak of A always precedes the peak of the R wave of the electrocardiogram In the dog a similar relationship can be observed

The onset of A is from 0 08" to 0 10" before the first sound, and the latter starts at about the middle of the descending branch of this wave (Fig 16)

Fig 16—Patterns of pressure of right atrium in normal subjects
(A) 5 year old girl
(B) 22 year old girl

If the phonocardiogram of the subject shows a fourth (atrial) sound, it is practically simultaneous with the peak of the A wave

The electrokymogram of the right atrium shows a small negative wave (decrease of volume) which takes place during the positive A wave of the pressure tracing (increase of pressure) *Both are expressions of the atrial contraction* (Fig 1)

Systolic Events

At the beginning of ventricular systole, a small notch in the pressure tracing reveals the closure of the tricuspid valve Instead of a notch there may be only a slower drop of the pressure curve or even a second (small) peak *during* the first sound We suggest that this notch or peak be represented by the symbol AV (atrioventricular) since the letter C frequently employed by others was originally used to describe the C wave

of the venous tracing and C stands for *carotid* according to the description of Mackenzie AV falls during the segment RS of the electrocardiogram, or at the peak of S

Another small, positive wave may occur later, *after* the end of the first sound (or during the end of that sound, if it is abnormally prolonged) This systolic notch is simultaneous with the rise of pressure in the aorta and is probably due to shaking of the right atrial wall by the ascending aorta This notch which can be called C is far from being constant

Most of the ejection phase is accompanied by a *decrease in pressure* which is due to lowering of the A V floor and the tricuspid valve (page 5 and Fig 5) as a result of the powerful pull by the ventricular septum and the right ventricular wall It should be called *systolic collapse*

Diastolic Wave

The ascending branch of the *systolic collapse* rises gently during the last part of ejection on account of abundant venous inflow into the atrium A new peak is reached from 0 04" to 0 07" *after* the main vibration of the second sound, at the time of the *opening* of the tricuspid valve (Fig 10) It is apparent that this opening causes a sudden change in pressure in the atrium by "removing the bottom of the chamber This peak may be called the V wave The peak of V is usually lower than that of A but may be equal to it and is never higher in normal individuals The peak of the V wave *follows* the end of the T wave of the ECG

SUPERIOR VENA CAVA

The pressure tracing of the superior cava is similar to that of the right atrium (Fig 17 B, C) Superimposed tracings show however, certain differences between RA and cavae (Fig 18)

(a) A slight delay of the A and V waves due to the time necessary for their transmission from the atrium to the cava

(b) A prominent C wave which is probably due to a systolic impact of the ascending aorta on the cava

(c) The possible lack of a separate notch for the A V wave

INFERIOR VENA CAVA

The pressure tracing of the inferior cava is similar to that of the superior cava However the peak of A is frequently more delayed than in the latter so that it may fall at the beginning of the first sound There may be no evidence of the notches AV and C at the beginning of systole (Fig 17 A) The delay between right atrial and inferior caval waves is greater than for the superior cava (Fig 18)

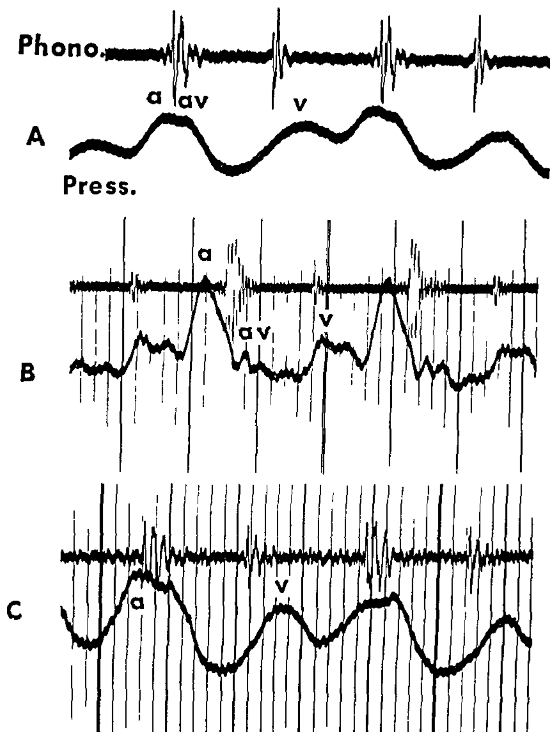


Fig 17—Patterns of pressure of the venae cavae in normal subjects
(A) 5 yr old child—inferior cava
(B) 14 yr old girl—superior cava
(C) 22 yr old girl—superior cava
a = presystolic wave v = early diastolic wave

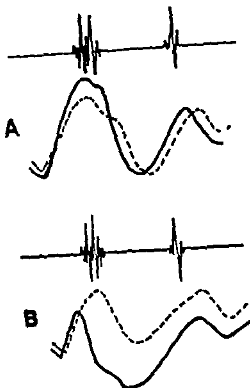


Fig 18—Relationship of caval to atrial pressure in normal subject (from original tracings)
(A) Continuous line = RA Stippled line = SVC
(B) Continuous line = RA Stippled line = IVC

LEFT ATRIUM

The left atrium is easily entered by retrograde arterial catheterization in the dog In man direct puncture appears to be a safer procedure (page 26)

The pressure tracing *of the dog* (Fig 3 A) shows a rapid often diphasic, presystolic wave (A wave) starting about 0 06″ before the beginning of the first sound a tall notch during the first sound (it should be called AV), a systolic collapse, and a high V wave

Left atrial patterns have been recorded in cases of atrial septal defect by Cournand and co-workers and by Nahas et al Direct puncture after thoracotomy allowed Wynn et al to study these pulses in normal subjects Later Facquet et al Allison and Linden, and Epps and Adler studied the left atrial pulse by transbronchial puncture while Bjoerk and Kent et al studied it by transthoracic puncture

We have recorded left atrial pressure pulses in *several normal dogs* The usual waves are the following

(a) The presystolic A wave of the left atrium is steep and tall and follows the A wave of the right atrium It may start with a small dip

(b) There is an extremely small notch during the first sound This notch which is caused by the closure of the mitral valve should not be called C (carotid) but rather AV (atrioventricular) like a similar notch found in the pressure tracing of the right atrium However, it is frequently absent

(c) There is a systolic collapse during ventricular systole

(d) Then one can see a gradual rise and then a tall V wave which coincides with the early diastolic opening of the mitral valve and follows by 0 05"–0 08" the main vibration of the second heart sound *

We have obtained pressure tracings from the left atrium in *2 infants* with no evidence of left to right or right to left shunt by passing the catheter through a patent foramen ovale (or small atrial septal defect) Also, we have recorded pressure tracings from the left atrium in *5 adults* having no evidence of mitral lesion or ventricular failure, through per cutaneous puncture of the left atrium, and found the following pattern (Fig 19) There is a peaked *A wave* followed by an *AV notch*, correspond

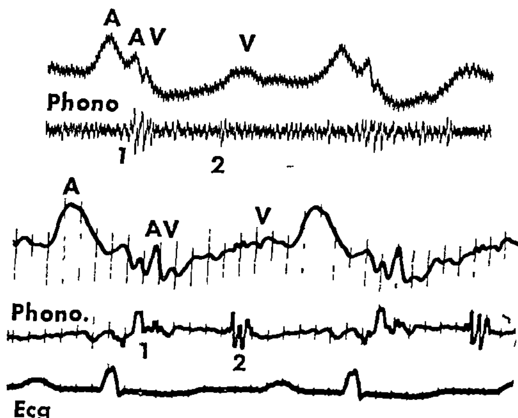


Fig 19—Pressure tracings of the left atrium (transthoracic approach) in two subjects where left atrial and left ventricular pressures resulted normal The patterns can be considered as typically normal
Phono = external phonocardiograms
Ecg = electrocardiogram

* Strangely enough certain authors place this V wave in late systole This can be explained only by poor technique or poor timing (the ECG is inadequate for timing this wave)

ing to the closure of the mitral valve * There is a negative wave *SC* (Systolic Collapse) during early ventricular systole The pressure gradually rises until it reaches a peak 0 06 to 0 08 second after the main vibration of the second heart sound and then begins to drop This peak is called the V wave and is definitely in early diastole † Slow diastole (or diastasis) which should occur later was absent in some of these cases on account of tachycardia

VENTRICLES

Ventricular pressure tracings have been recorded for over a century in animals The plateau like pulse described by Chauveau and Marey in the horse was later recorded also in the dog However, the shorter duration of systole in the latter animal frequently caused this pulse to have a more rounded contour According to a classic description, the typical tracing has a rise during the tension period (first part of first sound) reaches its maximum level at or soon after the end of the first sound, remains level or gently slopes down during systole then starts to drop rapidly at the beginning of protodiastole (shortly before the second sound) The lowest level is reached some time after the second sound and at the beginning of opening of the A V valves After this rapid filling begins and a short phase of rapid rise occurs which is soon followed by an even course (Fig 2) Certain variations may occur but are not remarkable

In order to exclude the different contours arising from a method different from that employed in man the authors catheterized dogs using a method which is identical to that currently used for right heart catheterization in man

The ventricular tracings of dogs are reproduced in Fig 20 They are similar to classic tracings recorded with older type manometers

Right Ventricle

Right ventricular tracings from normal persons are presented in Fig 21 They have a diastolic pressure which is at or near zero (Table I) The pattern which corresponds to the theoretically correct profile of right ventricular pressure is presented in (B), a descending slope of the plateau being a common variation Other tracings may present minor abnormali

* Several authors call C wave the notch which can be seen at the beginning of the first heart sound This connotation used because of resemblance between an atrial tracing and a jugular vein tracing is incorrect The C wave of the jugular tracing is a systolic wave due to impact of the aortic pulse on the superior cava The AV notch on the other hand occurs earlier a the beginning of the isometric period of systole Certain jugular tracings may show both an AV and a C wave (Luisada) while a left atrial tracing can only show the first

† See footnote page 26

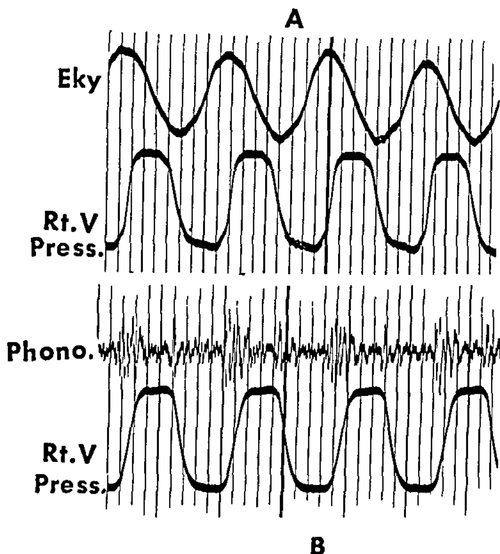


Fig 20—Ventricular patterns of normal dogs
(A) Electrokymogram and pressure tracing of right ventricle
(B) Phonocardiogram and pressure tracing of right ventricle

ties including slow rise and drop It is likely that minor overdamping (page 33), not sufficient to alter the levels of pressure, is the cause of these abnormalities It is important to note that, while the top of the plateau like pulse of the ventricle may have different variations, it never shows a progressive rise or a double rise (after the ascending slope), like some pathological tracings

Left Ventricle

The tracing of the left ventricle is similar to that of the right There is *a rectangular, plateau like wave during the entire systole* (Fig 22) Lesser amplification is necessary on account of higher pressure therefore, the small details of the tracing originating in valvular events are poorly visible or absent The drop in pressure which takes place shortly before

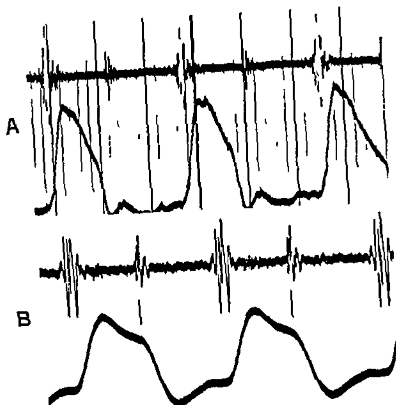


Fig 21—Right ventricular patterns of two subjects with normal pressures
(A) Boy of 14
(B) Girl of 5
Upper tracings = phonocardiograms

the second sound has a rapid descent the end point of which is at or near zero Early diastole is accompanied by a rapid rise of short duration (rapid inflow) next a slow slope or a steady course is visible in diastole The highest level of diastolic pressure is identical to the filling pressure of the left atrium

ARTERIES AND VEINS

Aorta, Brachial and Femoral Arteries

The arterial tracings recorded in animals for a long time, have been registered in man only in the last few years Katz et al first studied the aortic tracing by retrograde arterial catheterization The normal aortic tracing shows the changes of contour which are typical of the *central pulse* the anacrotic depression the peak, the incisura and the dicrotic wave Additional waves preceding the rise of the pulse have been explained as the result of presystolic atrial contraction causing a vibration of the aortic valve and by an early systolic bulging of the aortic leaflets during the tension period

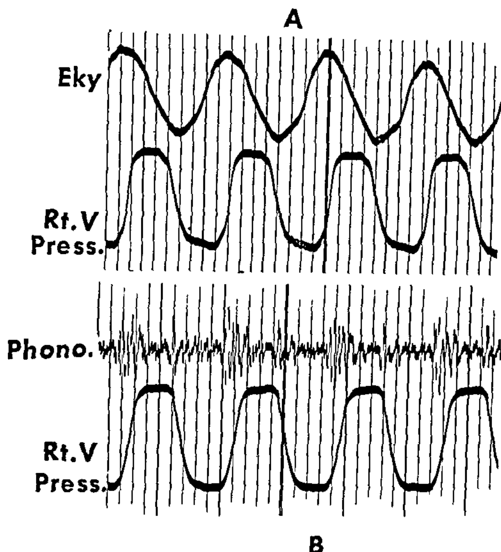


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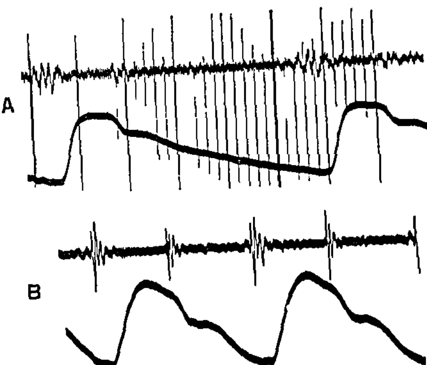


Fig 23—(A) The brachial pulse recorded by arterial catheterization in man Upper tracings phonocardiogram
(B) Pattern of the pulmonary artery Typical normal tracing in a girl of 5

distensibility of the pulmonary artery The anacrotic notch is usually not visible, the peak is rounded and falls at about $^2/_3$ of the ejection period the incisura is deep and rounded and is followed by a high dicrotic wave

In some tracings a high squarish wave or several slow vibrations can be observed in diastole These should be considered as artifacts, due to flopping of the catheter after the second sound and the effect of atrial and ventricular movements upon it Careful withdrawal and reinsertion of the catheter may cause the disappearance of these spurious waves

Pulmonary Stems

The pattern of the pressure pulses in the stems of the pulmonary artery is similar to that of the main artery and may be identical to it The peak of the main wave falls slightly later than in the pulmonary artery but still precedes the second sound by about one third of systole

Pulmonary Artery Wedge Tracing

(So called pulmonary "venous" tracing or pulmonary "capillary" tracing) This tracing is far more variable than any other because of the following technical difficulties

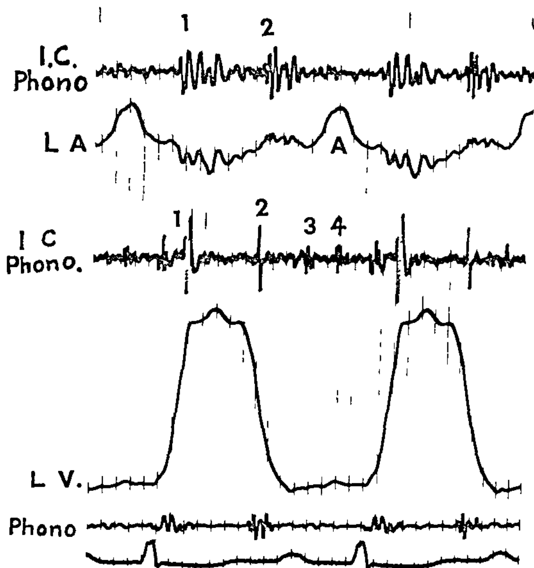


Fig 22—Intracardiac phonocardiograms (I C Phono) and pressure tracings of the left atrium and ventricle of a subject with normal atrial and ventricular pressures and patterns
1 2 3 4 = heart sounds
Phono = external phonocardiograms
The lowest tracing is an electrocardiogram (In order to save space the ECG and external phono of the left atrial tracing have been cut out and the atrial tracing has been exactly superimposed on the ventricular)

The brachial and femoral pulses, on the other hand, have the smoother contour of the *peripheral pulse* and show no evidence of an anacrotic depression (Fig 23 A)

Pulmonary Artery

This tracing is typical of an *arterial pulse* with a pattern resembling that of a *peripheral* pulse (Fig 23 B) The reason lies in the marked

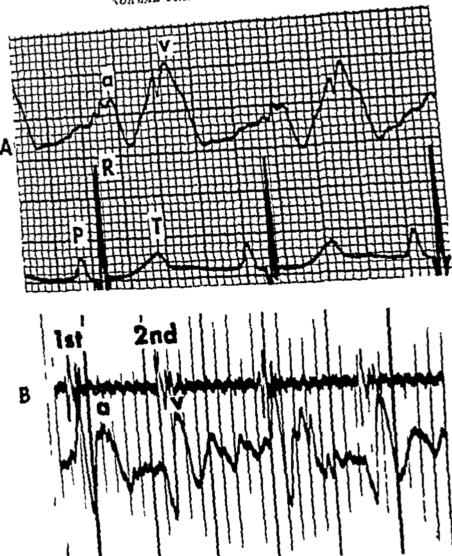


Fig 24– Pulmonary artery wedged tracings in two normal subjects
(A) direct writing apparatus lower tracing = electrocardiogram
(B) photographic recording upper tracing = phonocardiogram

the time of the *first sound* there is a diphasic oscillation usually of the positive negative type (Fig 24 A) which may be replaced by a mono phasic acute peak This notch is caused by a shaking of the catheter because of the double valvular movement which takes place at that time in the right heart Therefore it is the equivalent of *the first sound* Following this vibration there is a slow and smaller wave which takes place in early systole We have marked this as A (Fig 24 B), because it is probably caused by a delayed transmission of the left atrial A wave This seems proven by the fact that the wave takes place earlier in cases with a prolonged conduction time and is absent in cases with atrial

(a) Possible transmission of pulmonary arterial pulses *around the tip* of the wedged catheter

(b) Possible rapid transmission of left atrial pulses *through open arteriovenous thoroughfares*

(c) *Slow transmission* of left atrial pulses through the minute capillary vessels of the lungs

(d) *Shaking of the catheter* due to movements of the heart (through which it passes) and of the lung (to which it is fixed)

This explains the variability of pattern, not only from case to case, but even in the same subject if the catheter is slightly withdrawn and then wedged again

The recording of "wedge" pressure was described by Hellem, Dexter et al in 1948 They correctly attributed the mean pressure recorded by this technique to transmission of left atrial pressure However, they recognized that multiple artifacts frequently render the readings incorrect The pressure pulses recorded by firmly wedging the catheter into a small pulmonary arterial branch until it occludes the lumen were later studied and described as being similar to those of the left atrium (Hellem et al, Lagerloef and Werkoe, Soulié et al)

It has been stated that because of slow transmission of the waves from the left atrium to the pulmonary arterioles, the waves of the "wedge" arterial tracing are similar to those of the left atrium but delayed in time If this were so, a delay of not less than 0 05″ sec should be observed There should be a fixed shifting of both the A and the V wave The former would fall during or after the first sound (early systole) and the latter after about one third of diastole or even at mid diastole Actually, as will be shown later, only the A wave has a major shift, when present Later, it was suggested that a rapid transmission of pulse could occur through arteriovenous anastomoses * If this is true, it is understandable why the typical waves are *not* always present Closing the arteriovenous thoroughfares would prevent the rapid transmission of waves and cause a general damping by the capillary bed Only "mean" pressure would then be reliably recorded

We have recorded pulmonary artery "wedge" tracings in 4 normal dogs, in 8 normal subjects and in several clinical cases in which the pulses should not have been affected by the disease (small atrial septal defect, arterial hypertension, moderate pulmonary fibrosis) We have been impressed by the following facts

(a) The pattern is extremely variable and frequently consists of several small waves in systole and diastole

(b) There may be only minimal oscillations of pressure

(c) Occasional cases present a pattern resembling that described by Soulié et al

When the tracing is typical, the following details can be observed At

* This concept was advocated by Silber

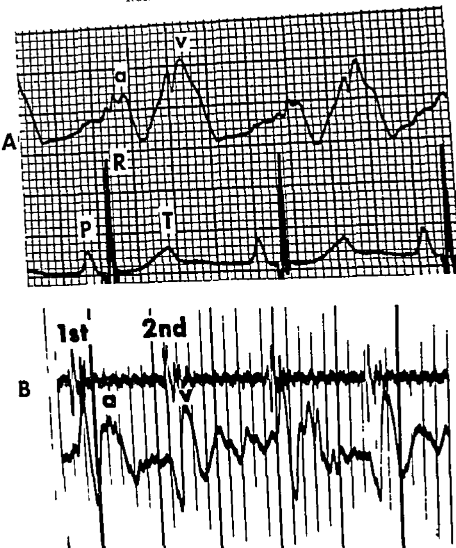


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fibrillation During the rest of systole, one can observe either a *systolic* collapse or a flat line With the *second sound*, there is a vibration which is usually of the negative positive type (Fig 24 B), this is immediately followed by a tall, positive wave coinciding with the opening of the mitral valve and which should be called the V wave (Fig 24 A and B)

It is interesting to note that, while the early systolic wave has a marked delay in comparison to the atrial contraction, the early diastolic wave has only a minimal delay over the opening of the valve This can be explained by *the fact that atrial contraction is weak and causes a wave* which moves *backwards* toward the capillary vessels of the lungs, while the opening of the A-V valve causes a sudden *forward* movement of a *long column of blood, from the capillaries to the left atrium*

Whenever a recognizable pattern is recorded, this may be studied and interpreted, otherwise, no such study can be made The most undesirable possibility is that of recording a pulse transmitted from the pulmonary artery, whenever this happens, it might be confused with a retrograde systolic wave due to mitral regurgitation However, such a wave is late systolic while that of the pulmonary artery is early systolic

Pulmonary Venous Wedge Tracing

(So called pulmonary arterial "capillary" tracing) This tracing can be recorded by left atrial catheterization via either the bronchus or the chest wall, and by firmly wedging the catheter into a pulmonary vein A similar tracing can be obtained by right heart catheterization if the catheter passes into the left atrium through an atrial septal defect The observed pattern is that of an arterial pulsation (Gensini et al)

CORONARY SINUS TRACING

The technique of catheterization of the coronary sinus was developed by Banfield et al and was studied by Bing et al The patterns of the



Fig 25—Tracing obtained by wedging the catheter in the coronary sinus Normal 5 yr old girl Upper tracing phonocardiogram

pressure pulses recorded by wedging the catheter in the coronary sinus, or by having it in the same vessel without obstructing the flow were studied by Read et al They describe a venous type of tracing if the tip is not obstructing the flow, with no evident C wave and a higher pressure than that of the right atrium They also describe a "ventricular" pattern if the catheter is obstructing the flow by occluding the opening

In one ca e, a child of 5 subsequently proved normal, we recorded the coronary sinus pattern with the catheter wedged in the vessel (Fig 25) The tracing had the following characteristics a drop in pressure in late pre systole and early systole, a sharp rise in pressure during early systole and a plateau which continues through the rest of systole *plus* most of diastole

No other known tracing has a similar pattern The form of this tracing may be explained by the systolic squeezing of the capillaries of the ventricular wall plus the well known diastolic flow through the capillary bed

CHAPTER FOUR

Normal Pressures of the Cardiac Chambers and Large Vessels

All pressure measurements should be based on the use of *the antero posterior mid chest level as a zero reference line* (Chapter two, zero reference line) Any pressure higher than this zero reference line is *positive*, any pressure lower than this line is *negative*

The normal pressures in the right and left heart are presented in Fig 15 and Tables III, IV and V

ATRIAL PRESSURE MEASUREMENTS

The *mean pressure* of the *left* atrium is slightly higher than that of the *right* atrium We have recorded left atrial pressures in 5 subjects with no evidence of mitral lesion or left ventricular failure and no enlargement of the left atrium noted by x ray The left atrial *mean pressure* varied from 6 to 9 5 mm Hg with an average of 7 7 mm Hg The average atrial *diastolic pressure*, just prior to atrial contraction, was 7 1 mm Hg, the average A wave was 12 1 mm Hg, and the *average mean atrial pressure during ventricular systole* was 6 6 mm Hg It should be emphasized that, in subjects with no evidence of mitral lesion or left ventricular failure, the mean atrial pressure during ventricular systole is either identical to or slightly lower than atrial diastolic pressure This presents a special interest when discussing pressure measurements in cases with mitral lesions (Chapter five)

The A wave is the point of highest pressure during atrial contraction The *systolic collapse* (S C, X or systolic descent) is the lowest pressure level and occurs during ventricular systole The V wave is the point of highest pressure during early diastole The diastolic drop or Y descent normally corresponds to the lowest pressure during early diastole Then the pressure gradually increases until the following atrial contraction begins (phase of so called *diastasis* or passive diastole)

By convention, the so called *atrial diastolic pressure* is the pressure *just before atrial contraction* or at the end of diastasis, about 0 15 sec prior to the Q wave of the ECG In rapid ventricular rate with short diastoles, atrial diastolic pressure is difficult to determine because there is no diastasis and there is only one wave culminating with the peak of A A level of pressure which is half way between the lowest point Y and the highest point A may be arbitrarily considered as "atrial diastolic pressure" with a

possible error of 2 mm Hg in normal subjects *Atrial mean pressure* is measured by planimetric or electric integration methods Atrial mean pressure *during ventricular systole* is the mean pressure level, measured by planimetric integration, from the point AV (closure of the A V valve) to the peak of V (opening of the A V valve)

The mean pressure of the *right* atrium is higher than intrathoracic pressure but very close to atmospheric pressure In an individual lying down at rest, right atrial mean pressure varies between 0 and +6 mm Hg Respiration and cough affect atrial pressures Right atrial pressure may drop to −7 mm Hg during inspiration and rise to +8 in expiration It may rise to +60 mm Hg during coughing *Mean right atrial pressure* is from 2 5 to 4 5 mm Hg (Tables III and IV)

Right Ventricle

The normal right ventricle has a systolic pressure of from +14 to +30 and a diastolic pressure of from 0 to +5 mm Hg (Tables I and II) The average is +22 systolic over +4 diastolic Inspiration may lower the pressures to +15/−6 expiration may raise them to +30/+7 Cough may cause a tremendous rise so that figures of +80 have been recorded in systole

Left Ventricle

The normal left ventricle has a *systolic* pressure identical to that of the aorta The *late diastolic pressure* varied from 6 to 9 mm Hg in 5 subjects with an average of 7 mm Hg It was mentioned previously (Chapter two) that, in a highly underdamped pressure tracing, sounds and murmurs originating in the ventricle produce high frequency vibrations which are superimposed on the pressure tracing Thus, overshooting of systolic pressure (upward) and diastolic pressure (downward) occurs The highest point of the vibration may be even 10 to 20 per cent higher than its actual pressure level On the other hand, the pressure measurements of an overdamped pressure tracing give lower systolic and higher diastolic pressures than the correct readings therefore, reliable pressure measurements of the ventricles can be obtained only with a proper recording pressure device If one is using an underdamped transducer, able to record high frequency vibrations (for example, a P 23 D Statham able to record heart sounds) a line should be drawn about mid way between vibrations as shown in Fig 13 This line will probably approximate the actual pressure level The *systolic pressure* of the ventricle is the highest pressure level during ventricular systole (with the above reservations) The ventricular *early diastolic pressure* is the lowest pressure level during early diastole and corresponds to the point of opening the A V valve *Late diastolic pressure* is the pressure just beofre atrial

contraction (or soon after atrial contraction if there is a prolonged P R interval) The term "ventricular end diastolic pressure" should be omitted because it may correspond to the rise in pressure due to atrial contraction, therefore giving an erroneously high reading

Arterial Pressure Measurements

Reliable systolic and diastolic pressure measurements of the pulmonary and systemic arteries should also be based on properly recorded pressure tracings Great care should be used in reading the pressure tracings of the pulmonary artery Numerous high frequency vibrations are created by the sounds or murmurs originating in the heart and pulmonary artery, while artifacts may be due to movements of the catheter Not infrequently, the catheter tip is obstructed during the greater part of ventricular systole Then, the highest pressure corresponds to the dicrotic notch and not to the real systolic pressure In this instance, a correct measurement of systolic pressure of the pulmonary artery *cannot* be done * Such an obstructive phenomenon was not observed during recording of the systemic arterial pressure, probably because the catheter often reached the artery in a retrograde way and not through the ventricle Even when the catheter was passed through the left atrium and ventricle into the aorta, it was a softer catheter with different physical properties from those of the catheter used in right heart catheterization In regard to the high frequency vibrations, which may be superimposed on the pressure tracings, a line drawn about midway between their extremes should give the correct pressure level

Systolic pressure is the highest pressure level of the artery during systole, *diastolic pressure* is the lowest diastolic pressure level *Mean arterial pressure*, used for the calculation of pulmonary and systemic resistances, should be measured in a routine fashion by planimetric or electric integration methods

Pulmonary Artery

The *main pulmonary artery* has a *systolic* pressure which should be very similar to, though possibly slightly lower than, that of the right ventricle More marked differences may be due to technical reasons (different intrathoracic pressure at different times of recording) A slight difference in pressure, not exceeding 5 mm Hg, is to be expected between the two chambers because part of the kinetic energy of the right ventricle is absorbed by the distensible wall of the artery in systole, and returned in diastole A somewhat larger drop in systolic pressure (up to 20 mm Hg) was said to occur in cases with increased flow and dilatation of

* Acceptance of this erroneous systolic pressure would cause the finding of a non existant pressure gradient between the right ventricle and pulmonary artery

the pulmonary artery * A systolic pressure between +14 and +26 should be considered normal in apnea while +10 and +30 should be the extremes of norm during respiration *Diastolic* pressure is markedly higher than that found in the right ventricle, due to the elastic recoil of the arterial wall, and may vary between +5 and +13 *Mean* pulmonary arterial pressure is between +11 and +17, the average being 14 (Tables III and IV)

Pulmonary Stems

The pressures are similar to those of the main artery However, here we have encountered more marked respiratory variations

Pulmonary artery wedge pressure (So called *pulmonary capillary venous pressure* or *indirect left atrial pressure*)

Like in the pulmonary or ventricular pressure tracings, numerous artifacts and high frequency sound vibrations may be superimposed on the main waves of the wedge pressure tracing These artifacts and vibrations can be eliminated or alleviated by slightly withdrawing the catheter (without dislodging it from the pulmonary arterial branch) or through the application of proper damping Pressure measurements of this pressure tracing are similar to those of atrial pressure tracings In a pullback maneuver from the pulmonary artery wedge position to the main pulmonary artery, a definite rise of both the systolic and diastolic pressures is usually observed (Fig 13 C) According to most authors, this pressure should be between 7 and 13 mm Hg and should present only small variations in coincidence with the cardiac cycle We have studied pulmonary artery wedge pressures and left atrial pressures in several subjects with an apparently normal left heart The mean pressures were identical to those of the left atrium and the levels of the waves also were similar to those of the left atrium the slight differences being within the limits of technical errors It is *unlikely that* the pulmonary artery wedge pressure actually represents the critical closing pressure of the pulmonary arterioles (Burton) and that whenever large systolic waves are seen the pressure readings should be always considered unreliable (too high) on account of leakage around the tip of the catheter † If pure oxygenated blood is aspirated when smaller catheters No 5 or 6 are used, the pressure reading should be considered correct

We have *recorded* cardiac and pulmonic pressures in 7 normal individuals between the ages of 5 and 48 years The data are reported in Table III Our readings show the various levels of pressure which

* This was not true in the cases with dilated pulmonary artery which were observed in our laboratory Artifacts may explain the apparent difference in pressure (see page 141)

† Obviously leakage may account for some of these readings Then the oxygen content of the blood is lower then in the arterial blood

TABLE III *Cardiovascular Pressures in Normal Humans (Luisada and Liu)*

No	Age	Sup Cava			Inf Cava			Right Atrium				Right Ventricle			Main P A			P A Stem		P A Wedge
		A	S C	V	A	S C	V	A	S C	V	Mean	Syst	Diastolic Beg	Diastolic Late	Syst	Diast	Mean	Syst	Diast	
1	5	—	—	—	—	—	—	5-7	3-4	4 5	4	25	0-3	6-8	15-26	5-12	16	—		—
2	20	—	—	—	—	—	—	7	4	8	3 5	14-16	0-3	3-5	14-15	8-10	11	10-16	5-10	—
3	14	6	1-2	6	—	—	—	5	0-1	4	3 5	20-25	−1 +1	2-4	20-25	6-10	13	—	—	8
4	5	—	—	—	—	—	—	—	—	—	—	28-30	0-2	3-5	24-26	8-11	17	21-26	8-11	11
5	48	7	1	4	7	1	4	7	0	3	6	22-25	0-2	5-7	—	—	—	—	—	—
6	12	—	—	—	5	1	4	4 5	−2	2 5	2 5	28	0	3	21	10	16	—	—	12 5
7	23	—	—	—	—	—	—	4 5	2 5	4 5	3 5	20	2	4	20	10	14	—	—	8
8	26	3 6	1 8	2 9	—	—	—	4 1	1 4	3 0	2 6	19	0 7	2 7	17 4	7 8	11	17	8 0	6
9	5	—	—	—	—	—	—	7 5	1 7	4 5	4 1	23	2 6	4 4	23	12 8	15	23	13	7
10	4	—	—	—	—	—	—	6 6	1 8	4 7	4 4	20	0	4	20	7 4	13	—	—	7 4
11	9	—	—	—	6 2	2 9	5 4	6 2	1 5	5 0	4 2	22	0	4	20	8	14	—	—	—

S C = Systolic Collapse

TABLE IV *Average Cardiovascular Pressures in Man*

	Right Atrium				Right Ventricle			Pulmonary Artery			P A Wedge Pressure	Left Atrium					
	A	S C	V	Mean	Syst	Diastolic Early	Diastolic Late	Syst	Diast	Mean		A	V	S C	Diast.	Mean systole	Mean
Fowler et al	2 5-7	0-5	2-7 5	1-4 5	19-31 5	—	2-6	1 6-29	5-13	10-18	4 5-13	—	—	—	—	—	—
Luisada and Liu	5 9	1 5	4 5	3 8	22 1	+1	4 1	21	9 1	14	8 5	12 1	8 2	4 5	7 1	6 6	7 7
Wynn et al	—	—	—	—	—	—	—	—	—	—	—	2-8	1-9	—	—	—	—

S C = Systolic Collapse

TABLE V *Pressures of the Left Heart in subjects with normal mitral valve and no evidence of left ventricular failure*

No	Age	Left Atrium							LV	
		A	AV	S C	V	Mean systole	Diast.	Mean	Syst.	Late diast.
1	32	12 6	14 7	3 7	10	7 0	6 9	8 3	148	7 0
2	36	8	7 0	4	7	5 8	6 2	6 0	97	6 2
3	o8	15	12 5	6	10	8 0	9 0	9 5	100	6-9
4	40	13	—	4	5	5 0	6 0	7 0	95	6
5	37	12	6 8	4 6	8 7	7 0	7 4	7 6	112	7 4
Average		12 1	8 2	4 5	8 2	6 6	7 1	7 7	110	7 0

can be found at different moments of the cardiac cycle and with different re^piratory phases

Fig 12 shows the ab^ence of a systolic gradient in the pullback maneuver from aorta to left ventricle, and the ab^ence of a diastolic gradient in the pullback from left \entricle to left atrium

CHAPTER FIVE

Intracardiac Phonocardiography

ANIMAL EXPERIMENTS

Records were obtained in 20 dogs by either right heart catheteriza tion (via the right jugular vein) or left heart catheterization (via the left femoral artery) (Figs 26–29) Catheters No 7, 100 cm long were employed In order to test its possibility, a thin catheter* was also used *Either the right or the left ventricular chamber was penetrated with a* No 18 thin walled needle (through the chest wall or with open chest) The thin catheter was then passed through the needle Further open chest experiments compared right and left atrial and ventricular tracings with the catheter passing either through one or more of the valves or directly through perforation of the atrial or ventricular wall The purpose was to test whether the valve leaflets were the source of vibrations by hitting the catheter

Sources and Relative Value of Sonic Frequencies

Vibrations of sonic frequencies were recorded from all four cardiac chambers, the superior cava, the pulmonary artery, by wedging the catheter in a small pulmonary vessel, and from the ascending and descending aorta (Figs 26–29)

The intensity of vibrations varied considerably according to the source *and increased in the following order superior cava, right atrium,* right ventricle and pulmonary artery, small pulmonary vessel and left atrium, left ventricle, it decreased from the left ventricle to the ascending aorta, and then sharply to the descending aorta

The vibrations obtained through the thin polyethylene catheter were slightly more damped but still clearly observable Check was made that no air bubble, blood clot or leakage existed in the system

Vibrations were observed in the frequency ranges 30 to 110, 60 to 110, 60 to 250 and 60 to 500 Occasionally, the highest range could not be used because of high frequency electric noise The vibrations were from 4 to 6 times larger in the left ventricle than in the right and from 2 to 3 times larger in the right ventricle than in the right atrium

Timing of the vibrations The tracings revealed the constant existence of two groups of vibrations grossly coinciding with the beginning and

* A polyethylene catheter size PE 50 50 to 60 cm long having an inner diameter of 0 57 mm was used

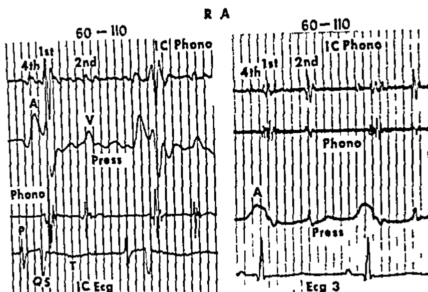


Fig 26—Intracardiac phonocardiograms from the right atrium of dogs
I C phono = intracardiac phonocardiograms
Press = pressure tracing
Phono = external phonocardiogram (at apex)
I C ECG = intracardiac electrocardiogram
ECG = electrocardiogram in lead 2

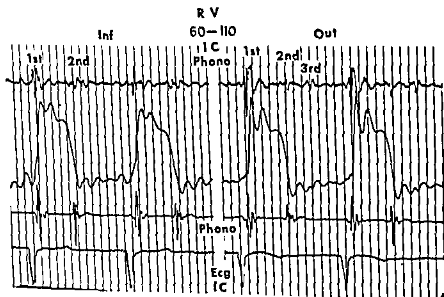


Fig 27—Intracardiac phonocardiograms from the right ventricle of dogs
Inf = inflow tract
Out = outflow tract
Other symbols as in Fig 26

TABLE VI *Timing of the Sonic Frequencies in Comparison with the External Apical Phonocardiogram*

No exp	Chamber	Delay of beginning of 1st sd over same phase of apical sd	Delay of main vibrat of 2nd sd over same phase of apical sd	3rd sd	4th sd	Opening sound of A V valve	Remarks
3	Superior cava	0 05–0 06	0 02	—	—	—	Vibrations often of low pitch
4	Right atrium	0 04	0 03	Good in all	Good in all	—	Vibrations often of low pitch
5	Right ventricle	0 03–0 04	0 02–0 04	Good in 3	Good in 2	—	Vibrations sometimes of low pitch
4	Pulmonary artery	0 03–0 05	0 02–0 03	Good in 1	Good in 2	—	—
3	Wedge PA tracing (LA)	0 02	0 01–0 02	Good in all	Good	—	—
3	*Left atrium*	*0–0 02*	*0–0 01*	—	Good in 1	Good in 1	—
7	Left ventricle	0 01–0 03	0–0 02	Good in 3	—	—	—
5	Ascending aorta	0 01–0 04	0–0 03	—	—	—	—
3	Descending aorta	0 05–0 06	0 03	—	—	—	—

the end of ventricular systole The timing of vibrations in regard to an external phonocardiogram varied according to the chamber in which the catheter was placed, as shown by Table VI In regard to the pressure tracings, the timing was always accurate

Diastolic extra sounds were also recorded in some of the tracings in coincidence with either the rapid diastolic filling phase (third sound) or the presystolic atrial contraction (fourth sound) (*Table VI*) Additional vibrations, not coinciding with the cardiac sounds, were found at times during ventricular systole, occasionally also during ventricular diastole, but only in the band 60 to 500

The response of the pressure transducer which was used (P 23 D) is such that there is a flat response up to 74 cycles/sec Higher frequency may still be recorded though attenuated in amplitude The resonant frequency of this strain gage is 280 cycles/sec Therefore vibrations in that range may be exaggerated in amplitude by this inherent property of the gage This might explain the occasional observation of a series of regular, high frequency vibrations within the ventricles of dogs during systole It was considered possible that they represented the exaggeration of vibrations of the chordae

A theoretical possibility, which was considered, is that by using a

system of triple differentiation, a record of *the rate* of rapid changes in pressure is obtained and not one of the rapid vibrations of sonic frequency Actually, diastolic vibrations were recorded while a simultaneous pressure tracing showed only minor and slow vibrations On the other hand, *whenever a sudden change in pressure takes place in a closed chamber, sonic vibrations necessarily arise, so that one tracing is equivalent to the other*

Several physiological tests excluded that the vibrations were due to impact of the valves on the catheter Hitting the tip of the catheter by the contracting ventricular walls or by the cardiac valves was usually revealed by artifacts, both in the pressure tracing and in the differentiated tracing Usually a slight withdrawal of the catheter led to disappearance of such extraneous vibrations Therefore, these possible mechanisms were also excluded

At this stage it was hypothetically accepted that certain vibrations were caused by and coincided with the heart sounds As the heart is a muscular organ it would be possible for sounds arising in one chamber or vessel to be transmitted to the others so that more or less the same vibrations would be recorded in all chambers This was disproven by the difference in phase magnitude duration and frequency of the vibrations recorded in the various chambers and large vessels It was apparent that the largest vibrations were taking place in the left ventricle and ascending aorta smaller vibrations in the left atrium and pulmonary vessels and smaller and lower pitched vibrations in the right ventricle Those of the right atrium superior cava and descending aorta were the smallest and at times could not be clearly recorded

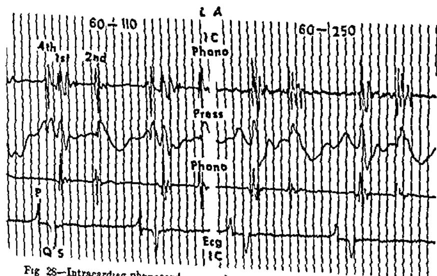


Fig 28—Intracardiac phonocardiograms from the left atrium of dogs (catheter) (Symbols as in Fig 26)

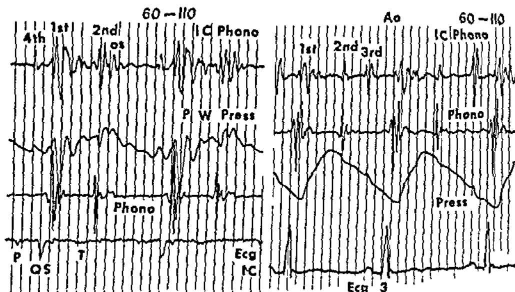


Fig 29—Intracardiac phonocardiograms from the pulmonary artery "wedge" position (at left) and the ascending aorta (at right) of a dog
(Symbols as in Fig 26)

The most likely interpretation is that *the recorded vibrations take place in the blood of the individual chambers or vessels as a result* of cardiac dynamics It is obvious that the contraction of each ventricle has the greatest importance by suddenly raising the pressure and causing the closure of the respective A V valve, the opening of the respective semilunar valve may also have importance by suddenly checking the rise in pressure

If the recorded vibrations are those of the blood, if they are caused indirectly by the muscular contraction and, more directly, by the motion of the cardiac valves, they are actually reproducing that part of the heart sounds which is arising in a particular chamber This, then, explains why the vibrations of the various chambers have a slightly different timing

The sound tracing recorded from the chest wall in our experiments is mainly a tracing of the left heart or at least has as a prominent feature the vibrations of the left heart This is explained first by the placing of the microphone on the left side of the sternum, and second by the preponderant part played by the left ventricle in cardiac dynamics The near coincidence of the *first and second left ventricular sound* with those recorded at the apex can be explained by these facts On the contrary the first and second right ventricular sounds are somewhat delayed because the right ventricle of the normal dog contracts slightly later (0 01–0 02 sec) than the left This was proven by recording two simultaneous ventricular pressure tracings The first sounds of the large arteries have often a slight delay over those of respective ventricles (0 01 to 0 02 sec) This can be explained by the absence of some of the initial vibra

tions of the first sound (mitral or tricuspid component) in the arterial tracing so that the beginning of the sound coincides with its second phase (opening of the aortic or pulmonic valve)

Occasionally, a split second sound was recorded in the pulmonary artery, more seldom in the ascending aorta Transmission of the vibration coinciding with the incisura of the aortic pulse to the pulmonary artery and vice versa explain this fact

A *fourth* (*atrial*) *sound* was recorded most often in the right atrium, less often in the left at times also in the right ventricle and pulmonary artery A *third sound* was recorded in both ventricles, especially in the outflow tracts and also in the right atrium

The vibrations of the descending aorta were sharply delayed over the cardiac sounds and were of a low pitch It is obvious that they represented vascular vibrations caused by the pulse wave

INTRACARDIAC PHONOCARDIOGRAPHY IN NORMAL SUBJECTS

Lewis and co workers found the following data in the right heart In all cases the first sound was recorded throughout the lesser circulation, and within the cavae The first and the third sounds were loudest in the ventricle, the second sound, in the pulmonary artery, the fourth sound in the atrium The major component of the first sound, recorded within the right ventricle started 0 06 to 0 07 sec after Q and coincided with the early part of pressure rise of the ventricle On the other hand the major component of the same sound, recorded within the pulmonary artery started 0 14 sec after the Q wave and was on the ascending limb of the pressure tracing of the same vessels A few small vibrations in the artery coincided with the larger vibrations in the ventricle It is obvious that the first group of vibrations was due to tricuspid closure, the second to pulmonic opening A systolic murmur was found in the pulmonary artery of normal persons It is likely that this was due to the characteristics of the microphone used by Lewis (extremely sensitive to high pitched vibrations) which recorded the minor turbulence of normal blood flow because this did not occur in the set up of the authors

Personal Observations

Two patients presenting no murmurs were submitted to both right and left catheterization The pressure tracings revealed no valvular lesions The intracardiac heart sound tracing recorded *in the left ventricle* of subject 1 reveal two components within the first heart sound (Fig 22) The first 2 to 3 vibrations is smaller and corresponds with the beginning of the rise of the left ventricular pressure it coincides with the closure of the mitral valve and follows the Q wave by 0 035 sec

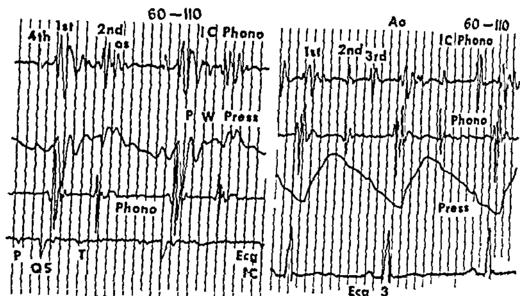


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CHAPTER SIX

Intracardiac Electrocardiogram

Right and left intracardiac electrocardiograms in *open chest experiments* were recorded by several authors following the studies of Thomas Lewis The first intracardiac tracings by means of *catheterization of the right heart* were recorded in dogs by Luisada et al in 1937 Clinical studies by means of right heart catheterization were made by Lenègre and Maurice in 1945, Hecht in 1946, Battro and Bidoggia, Sodi Pallares and co workers in 1947 and by Duchosal and co workers in 1948

Intracardiac electrocardiograms *of the left heart* were obtained accidentally during right heart catheterization in congenital heart disease when the catheter passed through a septal defect The first tracings were described by Sodi Pallares Duchosal and Kert More recently, Latour and Puech reported several tracings Subsequently, left cardiac electrocardiograms were studied through *retrograde aortic catheterization* A recent study in our laboratory (Luisada and Liu, 1957) was performed by using 5 per cent saline solution within a polyethylene catheter as conductor for the exploring electrode during *left heart catheterization* (Chapter two)

Analysis of the Waves

The *atrial complex* recorded *near the S A node* consists of a series of rapid deflections and a final deflection (*Ta*) The Ta wave represents the phase of atrial repolarization of the atria and is visible only in cases of A V block The interval from the beginning of P in the limb leads to the end of Ta measures from 0 34 to 0 42 sec but may be much longer

According to the position of the tip of the catheter various complexes can be recorded within *the right atrium* (Fig 30) These should be compared with epicardial complexes (Fig 31) At the root of the superior cava the ECG is similar to that of a VR However, at a slightly lower level, P is represented by a deep negative complex while a QS and an inverted T represents the processes of depolarization and repolarization of the ventricles At a lower level P has a small positive wave followed by a deep negative wave and if one moves toward the septum, the atrial complex has two equal positive negative phases P becomes positive near the inferior cava and near the tricuspid valve *The ventricular complex* is represented by a QS wave in the outward part of the atrium and

The second, larger component, having two vibrations, occurs at ⅔ of the rise of the ventricular pressure and seems to coincide with the opening of the aortic valve, it follows the Q wave by 0 105 sec The second heart sound, presenting a single vibration, coincides with the large vibration of the second sound recorded over the midprecordium (aortic component) It is, therefore, due to the closure of the aortic valve There is no murmur but a few minimal vibrations can be observed during systole The third heart sound, presenting one or two vibrations, occurs 0 13 to 0 14 sec after the second sound, in coincidence with the rapid filling of the left ventricle *The fourth heart sound, presenting a tri* phasic vibration, coincides with left atrial contraction

The intracardiac sound tracing recorded *in the left atrium* of the same subject (Fig 22) reveals vibrations of the *first sound* which coincide with the second group of vibrations recorded in the left ventricle (aortic component of the first sound), transmitted from the aorta, probably through contact of this vessel with the atrium The *second sound,* present ing a triphasic vibration, coincides with the aortic component of the second sound, recorded in the left ventricle The opening sound of the mitral valve presents a triphasic vibration and follows the first vibra tion of the second sound by 0 06 sec , it coincides with the V wave of the left atrial pressure tracing After the mitral opening sound, the re maining diastole is clear except for a few low frequency vibrations, coinciding with the atrial contraction, these vibrations may be termed "fourth heart sound" because they coincide with a larger, more rapid, and more distinct sound occurring within the ventricular cavity

Heart sounds recorded within the normal right ventricle and pul monary artery are presented in Fig 13 The intimate connection between ascending aorta and main pulmonary artery makes it possible to record both the aortic and the pulmonic components of the second sound from *within the pulmonary artery*, as well as the right ventricle (Fig 13)

is usually represented by a diphasic, positive negative, wave The different size of the positive versus the negative phase depends upon the proximity to the septum *The ventricular complex* is represented by qR at the highest part (Fig 30), rS near the left pulmonary veins, RS in the center and near the septum and QS near the mitral valve

In *the left ventricle* the typical pattern is represented by the typical complex, positive P QS ventricular complex, negative T The only exception is an occasional rS complex, recorded near the base of the septum

Mention should be made that, whenever the tip of the catheter touches the ventricular wall, an 'injury pattern' is recorded (Fig 32) This pattern is not revealed by a conventional ECG, as previously noted by

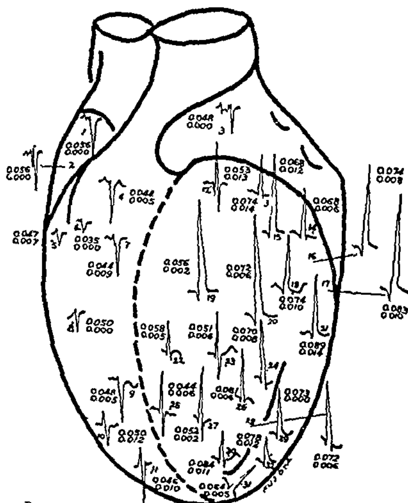


Fig 31—Patterns of epicardial electrocardiograms Compare with Fig 30 (Courtesy of Dr E Barbato)

by qR, and rSr waves near the septum (Fig 30) It is only near the tricuspid valve that a typical rS complex is recorded

In *the right ventricle*, a well known pattern is usually recorded = positive P, rS complex, negative T The only exception seems to be the occasional recording of an rSr complex near the base of the septum

In *the left atrium*, various patterns can be recorded *The atrial complex*

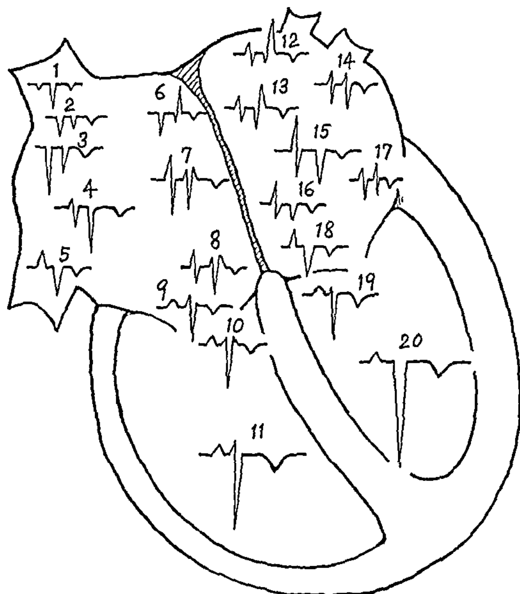


Fig 30—*The patterns of intracardiac electrocardiograms in the normal heart both from previous studies and observations of author*

(1) (2) (3) Superior cava (4) Center of right atrium (5) Inferior cava (6) (7) *(8) Medial points of right atrium* (8) (9), (10) *Above and below tricuspid valve* (11) Right ventricle (12) (14) Pulmonary veins (13) (16) Medial parts of left atrium (17) Lateral part of left atrium (18) (19) Above and below mitral valve (19) Unusual pattern occasionally recorded near the upper left part of ventricular septum (20) *Left ventricle*

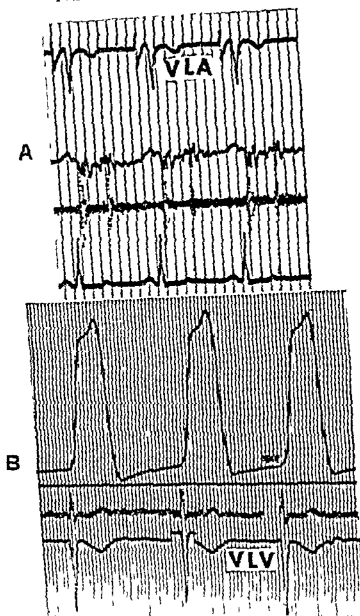


Fig 34—Normal patterns of ECG within the *left atrium* (A-top tracing) and *left ventricle* (B-lower tracing)

several authors particularly Sodi Pallares Withdrawal of the catheter is immediately followed by normalization of the tracing

Normal intracardiac electrocardiograms of the right heart are presented in Fig 33 those of the left heart are presented in Fig 34

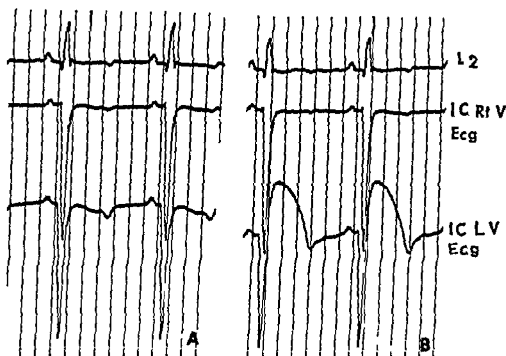


Fig 32—Electrocardiograms in a dog
From above lead 2
intracardiac V lead, right ventricle
intracardiac V lead left ventricle
Between (A) and (B) the tip of the LV catheter touched the ventricular wall originating the injury pattern of (B)

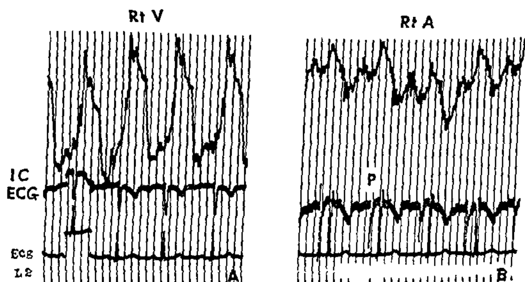


Fig 33—Intracardiac electrocardiograms of right heart
From above pressure tracing
intracardiac V lead
lead 2
(A) right ventricle
(B) right atrium

described a plateau pattern in the venae cavae and right atrium, due to tricuspid insufficiency, similar to that previously observed through clinical pulse tracings of the jugular veins and the liver

Theoretically, a systolic elevation of atrial pressure should be considered evidence of regurgitation through the incompetent A V valve, because regurgitation into the atrium occurs under a high ventricular systolic pressure (especially in the case of the left ventricle) This is particularly apparent because, in normal subjects left atrial mean pressure during ventricular systole is *not* higher than the pressure at mid or late diastole

The characteristics of either normal or abnormal human atrial distensibility are unknown In normal dogs, volume pressure changes of the left atrium have been found by Little to be linear only when the pressure was within normal limits (0 to 150 mm H_2O) When the normal limits of pressure are passed a slight increase in volume causes a great increase in pressure In other words, when atrial pressure exceeds the normal limits, increase in pressure is not linearly correlated with the increase of atrial volume

We have studied the border tracings of the left atrium by *electrokymography* (left atrial volume curve) and the left atrial pressure pulse in the same patients and found that their patterns are usually similar (Figs 47 and 50) Unfortunately the electrokymogram of the atrium cannot give at present the exact value of *changes of atrial volume* The expansion caused by regurgitation occurs under a high ventricular systolic pressure It is a pressure volume pressure change which cannot be determined as yet The systolic elevation (insufficiency wave) above the diastolic pressure level may not increase in a linear fashion in regard to the volume of regurgitant blood The level of mean atrial pressure during ventricular systole gives a more reliable evaluation of the regurgitation than the absolute value of the regurgitant wave Therefore, the *absolute height* of the regurgitant wave should not be entirely attributed to the result of insufficiency unless stenosis of the A V valve or resistance to flow (as in myocardial fibrosis or constrictive pericarditis, etc) can be ruled out In the presence of stenosis, only *the difference in level* between systole and diastole (mean systolic elevation) should be taken into consideration

Semilunar valves A low diastolic pressure and a wide pulse pressure are usually accepted as evidence of aortic regurgitation in syphilitic or rheumatic heart diseases However, it is not uncommon to find both a normal diastolic pressure and a normal pulse pressure in adults with clinical evidence of aortic insufficiency and stenosis It has been pointed out (Liu and Lima) that it is difficult to identify the presence of pulmonic insufficiency, because diastolic pressure of the pulmonary artery is already very low in normal subjects Unless the diastolic pressures of the pul

CHAPTER SEVEN

Abnormal Patterns

General Criteria of Interpretation of a Pressure Tracing

Evidence of dynamically significant valvular stenosis When the functional area of a valve is reduced to a critical level, valvular resistance to blood flow at rest occurs The pressure behind the stenotic valve increases in order to maintain a normal (or slightly reduced) flow Therefore, a *systolic pressure gradient* develops across a narrow semilunar valve and a *diastolic pressure gradient* develops across the narrow atrioventricular valve Since the square root of a pressure gradient is directly related to the amount of flow and is inversely related to the size of the opening, both the pressure gradient and the flow across the valve are important in order to evaluate the functional orifice of a stenotic valve (Chapter nine) Unfortunately, in rheumatic mitral or aortic stenosis, a various degree of concomitant insufficiency is frequently present The forward flow across the valve, obtained by Fick's principle, does not include the backward flow due to the insufficiency

The estimation of a regurgitant flow cannot be calculated unless an actual measurement of the valvular area has been done at autopsy or surgery Neither of them is of help for preoperative evaluation Although an indicator dilution curve was claimed to be of value for the calculation of regurgitation, its accuracy needs further proof With the existence of this unknown factor, it is apparent that a pressure gradient across a valve indicates only a dynamically significant degree of stenosis while the absolute value of the latter can be calculated only on the basis of the actual forward flow On the other hand, if no evidence of regurgitation is revealed by the pressure pulse pattern, as well as by *clinical examination* and other laboratory data, the greater the pressure gradient the smaller the valve, provided that cardiac output at rest is normal or only slightly reduced The authors wish to emphasize that the functional area of a stenotic valve should be correlated with the weight or body surface area of the patient

Evidence of dynamically significant valvular insufficiency

Atrioventricular valves In cases of right heart failure, systolic pulsations of the liver and of the jugular veins were often found and were interpreted as due to functional insufficiency of the tricuspid valve With moderate elevation of the right atrial pressure the obliteration of the systolic collapse or X descent has been described The authors have

monary artery and of the right ventricle are identical, the diagnosis of pulmonic insufficiency cannot be definitely made Intracardiac phonocardiograms taken in the left ventricle or the aorta near the valve may give certain information because a diastolic murmur is usually present in cases with insufficiency Still the insufficiency of the semilunar valves is more difficult to evaluate from pressure tracings than that of the A V valves except in severe cases

Superior and Inferior Venae Cavae

Several abnormal patterns can be observed

Tall A wave may be found in any case with high venous (and right atrial) pressures (Fig 35)

Double A wave is a curious phenomenon which still lacks an adequate explanation (Fig 35)

Multiple A waves may be observed in atrial flutter

Absence of A waves is observed in atrial fibrillation

Plateau pattern is typical of tricuspid insufficiency If this dynamic disturbance is functional or is unaccompanied by stenosis, the plateau resembles that of a ventricular tracing and its rise and fall coincide with the first and the second sounds If, on the contrary there also is valvular stenosis the plateau is a "late plateau" being delayed in its onset and termination or is irregular and taller at the end than at the beginning (Fig 35)

Right Atrial Patterns

The most common abnormal pattern is that caused by *increased pressure* in the right heart In such cases the A wave becomes tall and lasts longer According to Shepard the pressure of the A wave may reach 10 mm Hg or more Broadening of the wave may be such that it reaches the following A V wave and may practically fuse with it

Atrial fibrillation causes the disappearance of the A waves Multiple small atrial waves occurring in an irregular sequence may be present in some cases and reveal the contractions of bundles of fibers in the state of fibrillation

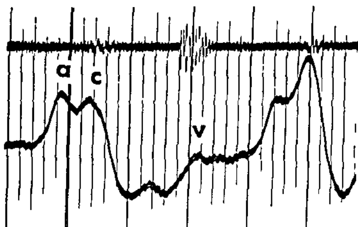
Constrictive pericarditis (Hansen Lin) and other conditions interfering with diastolic filling of the ventricle such as myocardial fibrosis,* amyloidosis (Dillon) and fibroelastosis (Lynfield) etc , are frequently accompanied by abnormal atrial patterns The most commonly found pattern consists of a high atrial pressure *high A wave* and an *early diastolic collapse* followed by a rapid increase of diastolic pressure

When tricuspid insufficiency is present the systolic collapse may be obliterated by an increase of atrial pressure during systole

* This pattern was observed by Robin and Burwell and by the authors







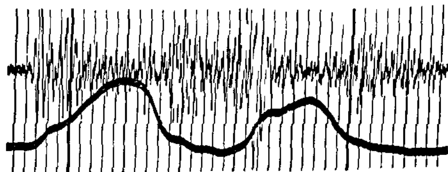


Fig 35—Abnormal caval patterns

(A) Tall A and C waves Cor pulmonale SVC
(B) Double A wave in Eisenmenger complex IVC
(C) A wave followed by tall C wave SVC
(D) Plateau pattern Rheumatic heart with mitral and tricuspid lesions IVC

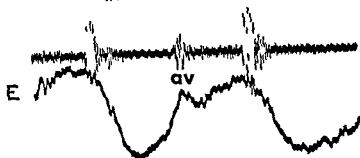


Fig 36 (*continued*)—Abnormal right atrial patterns
(E) Plateau pattern in tricuspid regurgitation (press 4/−3) The early peak of the plateau is marked as AV because of the similar mechanism with the AV wave of the normal tracing

Simultaneous right and left atrial pressures were recorded at the same sensitivity and midchest zero line by right and left heart catheterization in a patient of atrial septal defect A right to left shunt was demonstrated at the atrial level The pressure of the right atrium was higher than that of the left throughout the cardiac cycle (Fig 14)

Broad A waves (nodal or ventricular extrasystoles, nodal tachycardia, complete A V block) are either tall or normal They are always broad and flat and may present a double peak * We found this pattern in cases of pentalogy of Fallot cor pulmonale and Eisenmenger s complex (Fig 36) A similar pattern is reported by Shepard in pulmonic stenosis An exact explanation of this curious phenomenon is still lacking

Plateau pattern is typical or tricuspid regurgitation (Fig 36) In cases of atrial fibrillation the plateau is particularly apparent on account of the absence of the A wave This pattern can be found in cases of rheumatic heart with mitral stenosis and either relative or organic tricuspid insufficiency In rheumatic cases, with some degree of stenosis, it shows (a) a delay of the onset and end of the plateau over the first and second heart sounds respectively (*late plateau*), and (b) an increased gradient of pressure in diastole between right atrium and right ventricle

Ventricular Patterns

Abnormal ventricular patterns have been the object of systematic experimental investigations by Wiggers In severe *pericardial effusion*, the initial intraventricular tension rises almost proportionally with intrapericardial pressure but the volume of the ventricles in diastole is decreased due to the compression developed by the fluid As a result, smaller ventricular pulses are observed and the gradient of the pulse becomes slower

* Double peaked A wave has been found in the jugular tracings of patients with atrial septal defect by Gelfand Even though this pattern may be occasionally found it is not as constant as stated

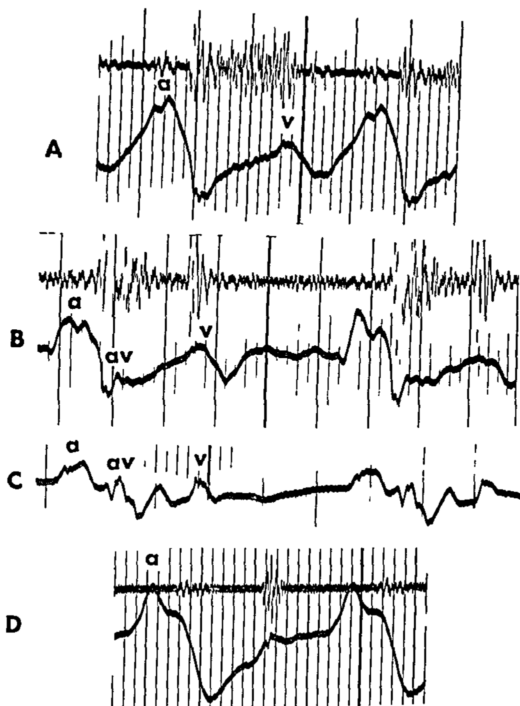


Fig 36—Abnormal right atrial patterns

(A) Tall double peaked A waves deep systolic collapse in pulmonic stenosis (press 6 5/0)
(B) Broad A waves in pentalogy of Fallot (mean press 6)
(C) Broad double peaked A waves in cor pulmonale (press 6/2 5)
(D) Broad A waves in Eisenmenger complex (press 7/1)

The immediate effect of *arterial hypertension* is a slightly longer isometric contraction and an ejection period which is so shortened that systole as a whole, becomes shorter The pressure rises to a higher peak, but a small amount of blood is retained at each beat, thus causing a greater initial tension In a later stage, initial tension is higher, the isometric gradient is steeper, isometric contraction decreases and the ejection phase becomes prolonged The pattern of the ventricular pulse is now changed and, instead of a rectangular plateau, there is a *more conical wave with a late peak*

In *coronary occlusion*, as shown by Orias the typical changes are a slower rise during the tension period without prolongation of this phase, a shorter period of ejection and a low, rounded peak The *systolic expansion* of the infarcted area, first demonstrated in animals by Wiggers and Tennant and later confirmed by roentgenkymographic and electrokymographic observations in man is largely responsible for the changes

In *pulmonic or aortic stenosis* the pulse wave of the affected ventricle becomes higher and more peaked When ejection is severely impeded, the ventricular contraction becomes nearly isometric with great rise in pressure and little ejection While in experimental aortic stenosis the rise in pressure may be tremendous in experimental pulmonic stenosis the pressure soon falls and the pulse wave is small and conical

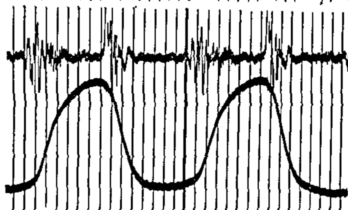
Right ventricular patterns In our series we have noted different right ventricular patterns which can be considered either *normal or borderline* They include (a) the typical plateau pattern (b) the conical pattern and (c) the pattern with descending slope

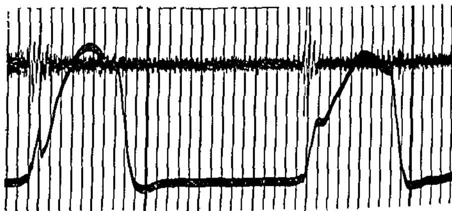
On the other hand two other patterns which seem to indicate a *definitely abnormal type of contraction* (Fig 37) are (a) the double peaked wave and (b) the slowly ascending or early notched plateau

These abnormal patterns were found in patients who had a high right ventricular systolic *pressure* Some also had an increase of right ventricular *diastolic* pressure all presented electrocardiographic evidence of right ventricular hypertrophy and ischemia (so called strain pattern) It was therefore concluded that these abnormal patterns reflected a condition of *mechanical strain* of the right ventricle

Two other abnormalities *of the right* ventricular tracing may be observed One of them consists of a *high diastolic pressure* (9 to 15 mm Hg) due to right heart failure This high pressure is noticeable at the end of diastole (*high filling pressure*) while the initial drop may be normal in appearance A correspondingly high right atrial pressure is always noted This high diastolic pressure was described by Mc Michael and by Harvey The other abnormality described by Hansen et al consists of *a sharp dip in early diastole* followed by a flat diastolic course (Fig 38 B) This pattern was originally described in *constrictive pericarditis* but can be







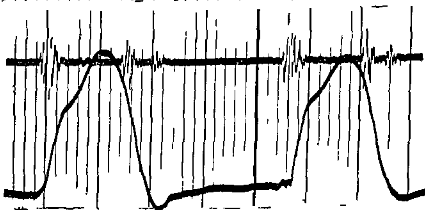


Fig 37—Patterns of ventricular strain

(A) Double peaked wave (press 50/−1+11)
(B) Slowly ascending wave (press 30/+3+6)
(C) Early notched plateau (press 71/+10)
(D) Early notched plateau (press 100/+10 +15)

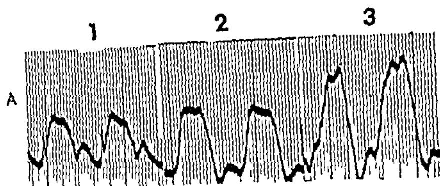


Fig 39—(A) Pull back from PA (1) to first part of infundibulum (2) and to main infundibulum (3) in a case with conical narrowing of last part of infundibulum plus infundibular stenosis of the right ventricle

Pulmonary Artery Patterns

The pulse of the pulmonary artery may present variants due to the *position of the peak* (Fig 41) This peak may occur either near the end of the first sound or during late systole As the position of the peak is determined by the ratio between pulmonary flow and right ventricular output an early peak logically indicates a free flow A late peak, on the other hand may be caused by *distal obstruction* (arteriolar, capillary or mitral), *weak right ventricle* or both

A second variation is due to the existence of a depression during the ascending branch of the pulse (*anacrotic depression*) An anacrotic depression in the aortic pulse is currently interpreted as being due to weakening of the left ventricle in the presence of severe hypertension or aortic stenosis It seems logical to explain in the same way the anacrotism of the pulmonary pulse if artifacts can be ruled out

The existence of multiple waves in systole a high dicrotic wave or a negative wave preceding the early systolic rise should be considered as artefacts On the other hand *absence of the dicrotic wave* (Fig 41) is noted only in cases of severe pulmonary hypertension

Severe pulmonic stenosis of the valvular type causes a complete distortion of the pattern (Fig 42) The ascending branch becomes slow and oblique and shows the inscription of several rapid vibrations which are the equivalent of the systolic thrill By analogy with events caused by aortic stenosis this could be called the "pulmonic shudder" As shown by Fig 42 there may be a late peak coinciding with a loud second sound (obviously transmitted from the aorta) while the second pulmonic sound is extremely weak and delayed

Pulmonary Artery Wedge Patterns

As already stated patterns recorded with the catheter firmly wedged into a small pulmonic branch present wide variations even in normal

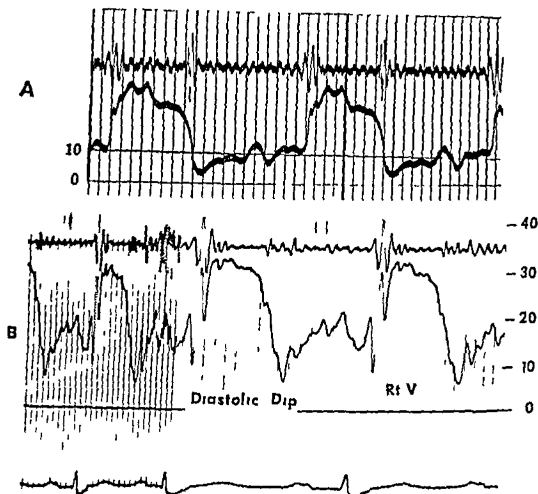


Fig 38—(A) Right ventricle High diastolic pressure in a case of initial right ventricular failure

(B) Diastolic dip of the right ventricle in a case of myocardial fibrosis with decrease of elasticity of the wall (same case as in Fig 53 B) The I C phonocardiogram shows a third heart sound (0 10–0 11 after closure of pulmonic valve)

found in other conditions impairing diastole, such as *endocardial fibroelastosis* and *myocardial amyloidosis*, as well as in *severe failure*

Right Infundibular Pattern

This pattern is never encountered unless there is a congenital subvalvular stenosis The latter is caused by a deformity of the ventricular septum which often creates a separate, small, infundibular chamber In such cases, the systolic pressure of the chamber is lower than that of the main right ventricle while the diastolic pressure is lower than that of the pulmonary artery and may be zero A pull back tracing from PA to *infundibulum* and from this chamber to RV clearly shows the differences between the various levels of pressure (Fig 39) The infundibular pattern has a slowly rising plateau during systole, and may be abruptly terminated by a peak in late systole (Fig 40)

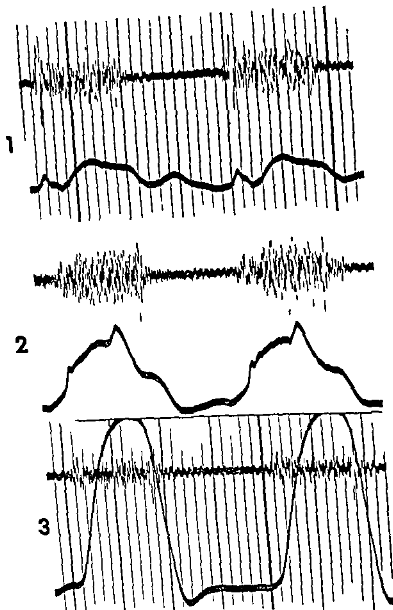


Fig 40—Pressure patterns in the case of Fig 39
(1) Pulmonary artery (press 24/18/16)
(2) Main infundibular chamber (press 35/−2 to +6)
(3) Right ventricle (press 82/−2 to +6)

subjects (page 53) In patients with heart disease, the same variations can be found Whenever two waves are observed (one during the first sound the other soon after the second) the tracing may be considered as representing the transmission of the left atrial pulse In general this pattern corresponds to a level of "wedged pressure which is within normal limits

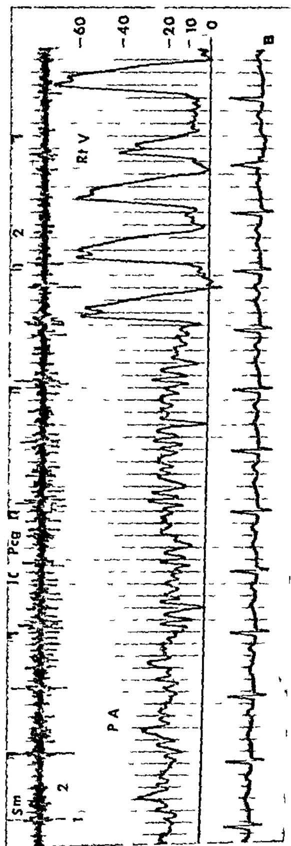


Fig 39 (*continued*)
(B) Pullback maneuver from PA to RV in a case with severe pulmonic stenosis of valvular type Intracardiac phonocardiogram

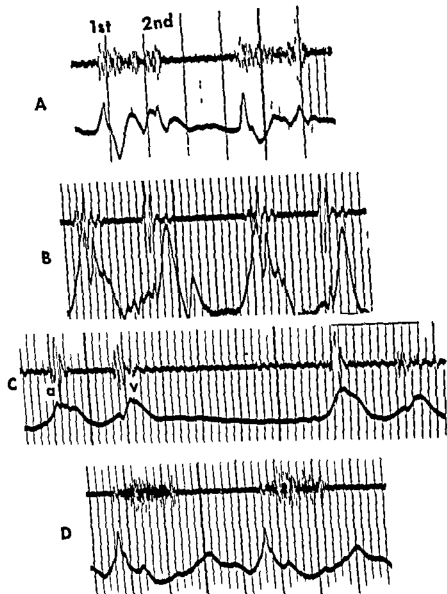


Fig 43—Normal patterns of wedged pulmonary artery pulses in cardiac cases Ventricular systole is between first and second sound
(A) Pulmonic stenosis and ventricular septal defect Mean pressure = 15
(B) Mitral stenosis plus tricuspid insufficiency (record taken one year after mitral valvotomy) Mean pressure = 13
(C) Mitral stenosis Mean pressure = 15
(D) Ventricular (and probably atrial) septal defects Mean pressure = 10

Mitral stenosis with high pulmonary venous and 'wedge' pressures (and no appreciable regurgitation) may be accompanied by a pattern of slowly rising pressure (Fig 44) On the other hand mitral stenosis plus regurgitation may be accompanied by *a new wave in late systole* (Fig 44)

A

B

a d

Fig 41—Pressure pulse of the pulmonary artery in patients with pulmonary hypertension
(A) Late peak no dicrotic wave (press 100/55)
(B) Anacrotic depression (a) and high dicrotic wave (d) (press 73/39) (See Fig 37 (C) for corresponding right ventricular pattern)

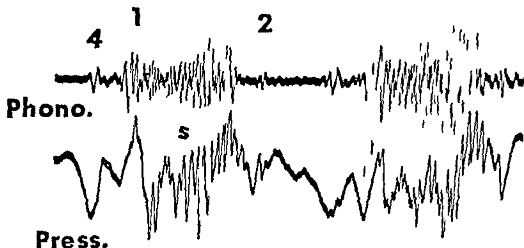


Fig 42—Phonocardiogram and pulmonary arterial pulse (press) in a case of severe pulmonic stenosis Note slow rise and pulmonic shudder (S)
(2) indicates weak second pulmonic sound
(4) is the fourth (atrial) sound

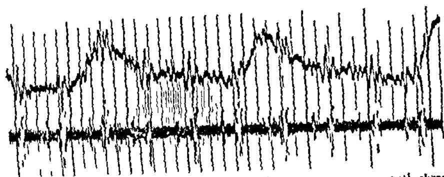


Fig 45—Severe respiratory variations of the PA wedge pressures in a case with chronic pulmonary disease

this wave is inconstant, so that, while its presence adds an interesting datum to the various signs of mitral regurgitation, its absence does not exclude this defect

Severe variations in the basic level of "wedge" pressure due to respiration are encountered in cases with obstruction of the capillary bed (Fig 45)

Abnormal Left Atrial Patterns

Haring Liu and Trace of this laboratory, made a comparative study of the left atrial electrokymogram left atrial pressure tracing, and pulmonary wedge pressure tracing in 32 dogs with experimental mitral lesions Electrocardiograms, phonocardiograms and autopsy observations were also made

It was ascertained that while mitral stenosis alone or with insufficiency raises the left atrial mean pressure and the pulmonary artery wedge pressure pure mitral insufficiency does so to a much lesser extent

It was determined that pure mitral stenosis, while raising the left atrial pressure during ventricular diastole, leaves the left atrial pattern relatively unchanged Taller A and V waves and a steeper rise during the second part of the systolic collapse were the only modifications

Pure mitral insufficiency was revealed by the substitution of a squarish plateau for the systolic collapse of the left atrial pattern (Fig 46) In general the rise of the plateau was rapid (*early plateau*) A more gradual rise occurred in 2 animals and was explained as due to incomplete compensation by the left ventricle in the early stages following surgery The pulmonary wedge pattern was found similar to the left atrial pattern in two animals with severe insufficiency however, the rise of the plateau was delayed in the former tracing (Fig 46)

In mitral insufficiency and stenosis the same abnormal patterns were found with the exception that the rise and fall of the plateau wave were gradual (late plateau) and that the diastolic pressure was higher than in normal animals

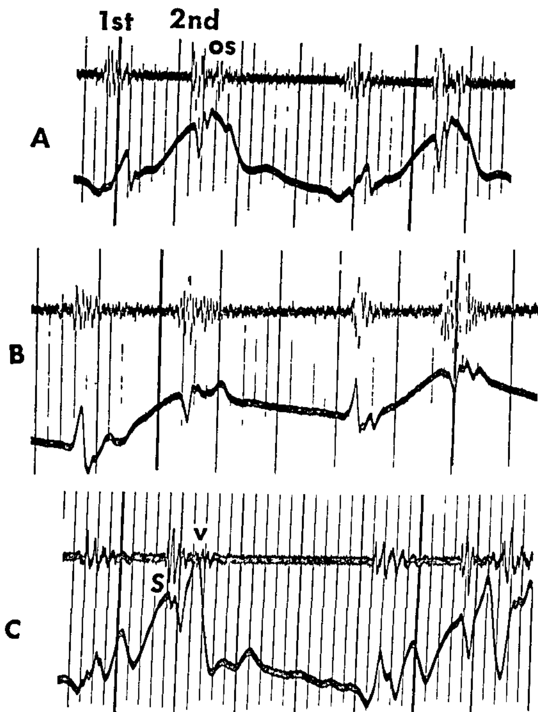


Fig 44—Mitral valve lesions There is an early rise of pressure during ventricular systole
(A) PA wedge = mean 30 (predominant mitral stenosis)
(B) PA wedge = mean 26 (predominant mitral stenosis)
(C) PA wedge = mean 28–30 In this tracing the wave (S) may be revealing some degree of mitral regurgitation

As pointed out originally by Gorlin et al, Lagerloef and Werkoe and Soulié et al, this wave is due to the transmission across the capillary bed of that regurgitant jet which penetrates the left atrium during ventricular systole because of mitral incompetence Unfortunately, the recording of

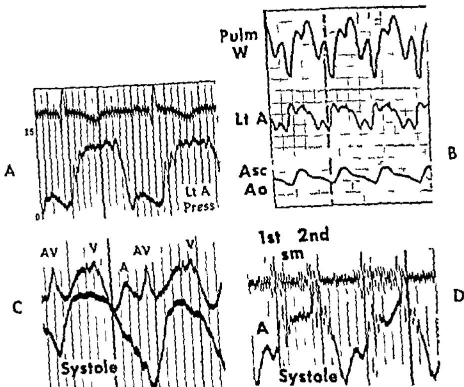


Fig 46—Tracings in dogs with experimental mitral insufficiency

(A) Electrocardiogram and pressure pattern of left atrium (rectangular plateau pattern)

(B) Simultaneously recorded tracings
Pulm W = pulmonary artery wedge pulse
Lt A = left atrial pulse
Asc Ao = pulse of ascending aorta
The wedge pulse is similar to that of the left atrium but delayed in time Both present a plateau pattern slightly distorted by superior position of the atrial usual waves

(C) Left atrial pressure pulse (above) and electrokymogram of left atrium (below) The pressure tracing rises more slowly during ventricular systole Thus only the EKY has a real plateau

(D) The same animal as in (C) studied two weeks later The insufficiency of the mitral valve has caused some degree of left ventricular failure The rise of left atrial diastolic pressure has caused a greater tension of the atrial wall Thus intraventricular pressure is more accurately transmitted and there is a plateau pattern
Above—phonocardiogram (external)
Below—pressure tracing of LA

of the left atrium during systole from the pulmonary veins (which have a very high pressure) is so rapid that it simulates the pattern of mitral regurgitation However this would account for only a minimal rise of mean pressure (2 mm Hg) above the late diastolic pressure during ventricular systole because the pulmonary veins do not act as a pump On

The electrokymograms of the left atrium were similar to the pressure pulses of this chamber (Fig 46) They revealed a plateau like wave in stead of the normal collapse during ventricular systole whenever there was regurgitation If no mitral stenosis was associated, the plateau was of an "early" type If there also was stenosis, the plateau was of a "late" type Pure mitral stenosis had no plateau In one instance, the pressure tracing rose more slowly than the electrokymogram tracing (Fig 46) This fact, due to elastic distention of the atrial wall, may be duplicated in clinical cases

The first abnormal left atrial pressure pulses in clinical cases were published by Munnell and Lam (open chest) followed by Cournand et al, Lagerloef and Werkoe, and Calazel, Bing et al (catheter passed through an atrial septal defect) Since, then, numerous authors have studied this pattern by direct puncture of the atrium either at thoracotomy or with a closed chest (transbronchial or transthoracic) The importance of such a pattern in rheumatic mitral patients is self evident

Unfortunately, inexact terminology, incorrect interpretation of some tracings and the division of cases on the basis of either clinical diagno sis or evaluation by the surgeon (both inadequate for a careful study) render evaluation and comparison of their data extremely difficult

As in the right heart, increased filling pressure of the left atrium is revealed by tall and broad A waves and by higher atrial pressure Atrial fibrillation causes the disappearance of the A wave

We have studied this type of tracing in 24 cases through left heart catheterization (Table VII) Decision on the type of hemodynamic dis turbance was based on the diastolic gradient and the systolic elevation

Mitral stenosis The hemodynamic diagnosis of mitral stenosis is based on the determination of the diastolic gradient

The studies of Braunwald et al have revealed that a "mitral block" results in a higher level of atrial pressure and, possibly, in an exaggeration of the A and V waves In such cases, filling of the atrium will take place more rapidly and will cause an *earlier rise of the tracing in late systole* Any other pattern should be interpreted as being caused by summation of the phenomena due to insufficiency (systolic elevation) with those due to stenosis (high diastolic pressure)

Mitral regurgitation Considerable confusion exists in the literature regarding evidence for mitral regurgitation The left atrial pressure tracing of normal individuals (5 cases observed by us) shows a gentle de pression during ventricular systole between AV and V As this is due to lowering of the A V floor by left ventricular pull it is possible that rigidity of the mitral leaflets decreases this depression either partly or totally, causing a flatter course of the tracing during ventricular systole

It has been said that, in patients with severe mitral stenosis, filling

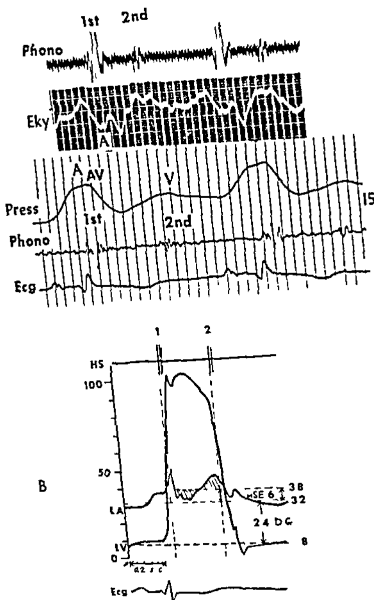


Fig 47—Case of nearly pure mitral stenosis
(A) From above—phonocardiogram (for timing)
electrokymogram of left atrium (normal pattern deep A wave)
pressure tracing of LA (tall A wave otherwise normal pattern)
phonocardiogram (external)
electrocardiogram
(B) Same case Superimposed left ventricular and left atrial tracings showing diastolic gradient across mitral valve (D G = 24 mm) and minimal systolic elevation (S E = 6 mm) (From Am J Card 1958)

TABLE VII *Tracings in 24 Cases Through Left Heart Catheterization*

No	Name	Age	Sex	Left Atrial Pressure (mm Hg)					Left Vent mm Hg		Stenosis Effect LA_d–LV_d mm Hg	Stenosis Index	Insufficiency Effect LA_m–LA_d mm Hg	Insuff index	I index / LA_d × 10	Ry/Ti	Ry/LA (mean)	M V R dynes sec cm	M V A sq cm	Remarks
				diast	A wave	systolic mean	I or IV wave	L A mean	syst	diast										
1	P A	37	F	25	35	27	30	27	94	6	19	4 36	2	1 41	5 6	1 00	1 08	205		No regurgitation felt
2	L S	43	M	21	34	22	26	24	106	8	16	4 0	2	1 41	6 7	0 81	0 87	253	0 81	No regurgitation felt
3	H E	42	M	29	32	30	34	32	147	12	17	4 13	1	1 0	3 4	0 89	0 91	—	—	No regurgitation felt
4	S L	30	F	22	30	23	25	25	106	8	14	3 74	1	1 0	4 5	1 00	1 00	—	—	No surgery
5	M M	37	F	27	32	36	42	32	107	4	23	4 8	9	3 0	11 1	1 4	1 94	275	0 71	No surgery
6	M R	27	F	32	36	39	45	38	106	4	28	5 3	7	2 65	8 3	2 1	2 50	288	0 72	Minimal insufficiency felt
7	D S	40	F	20	25	25	27	23	110	4	16	4 0	5	2 24	11 2	1 00	1 17	—	—	No regurgitation felt
8	B G	39	F	26	—	37	39	31	110	8	18	4 25	11	3 32	12 8	3 33	3 72	239	0 68	No regurgitation felt
9	B A	41	F	27	—	38	40	32	110	4	23	4 8	11	3 32	12 3	1 94	2 42	—	—	Moderate regurgitation felt
10	P M	33	M	26	34	33	36	30	108	6	20	4 47	9	3 00	11 5	1 34	1 82	199	0 97	No regurgitation felt
11	S S	39	M	22	—	28	30	26	130	6	16	4 00	8	2 83	12 9	1 61	2 12	199	0 86	No regurgitation felt
12	M C	35	F	19	—	29	30	24	92	9	10	3 16	10	3 16	16 6	0 67	0 64	133	1 25	No regurgitation felt tip of index finger
13	A V	54	F	19	—	27	28	24	105	8	11	3 32	9	3 00	15 8	1 44	1 72	142	0 96	No regurgitation
14	F G	41	F	20	—	28	34	24	125	7	13	3 61	8	2 83	14 1	1 00	1 35	—	—	No surgery
15	W I	40	M	14	—	20	23	17	120	4	10	3 16	6	2 45	17 5	2 27	2 28	128	1 16	No surgery
16	L W	46	M	19	—	22	23	21	106	11	8	2 83	3	1 75	9 2	0 97	1 32	97	1 25	Significant moderate regurgitation felt
17	R M	30	F	12	—	15	16	14	114	7	8	2 83	3	1 71	14 3	2 50	2 62	95	1 15	No surgery
18	S I	44	F	14	—	21	32	18	100	7	7	2 64	7	2 64	11 7	1 21	1 75	—	—	+ + regurgitation
19	C S	36	M	16	16	38	72	29	106	16	0	0	22	4 7	29 4	4 30	12 0	—	—	Autopsy no mitral stenosis dilated mitral ring
20	D D	33	F	21	—	36	49	30	90	20	1	1	15	3 88	18 5	3 80	5 6	—	—	No surgery
21	M G	39	F	14	—	35	44	26	150	14	0	0	21	4 59	32 8	5 7	6 8	—	—	No surgery
22	K B	21	F	6	13	12	14	10	120	6	0	0	6	2 45	40 8	5 5	8 1	—	—	No surgery
23	W L	47	F	14	—	22	29	20	115	9	5	2 24	8	2 83	20 2	3 1	4 4	57	1 66	No surgery
24	M P	—	M	12	—	17	19 4	14	109	11	5	2 24	5	2 24	19 0	4 05	4 4[illegible]	50	1 91	No surgery

Stenosis index = square root of LA_d–LV_d
Insufficiency index = square root of LA_m–LA_d
I index = insufficiency index

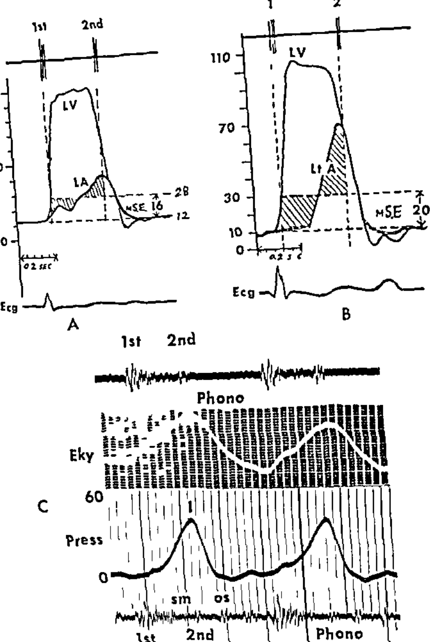


Fig 48—Three ca. es of mitral insufficiency and *no* stenosis

(A) Superimpo ed left ventricular and left atrial pressures in a case of medium severe insufficiency Slowly rising left atrial pressure during ventricular sys tole Systolic elevation = 16 mm

(B) Similar tracings in a case of extremely severe insufficiency Rapidly rising left atrial pressure culminating in a tall pointed I wave which coincides in time with the 2nd sound and precedes the time of opening of the mitral valve (V wave) (From Am J Card 1958)

(C) Other ca e of severe insufficiency
From above phonocardiogram
electrokymogram of left atrium (I wave)
pressure tracing of left atrium (I wave)
phonocardiogram
(From Am J Card 1958)



Fig 47 (continued)—Case of nearly pure mitral stenosis
(C) Other case of pure mitral stenosis Superimposed left ventricular and left atrial pressure tracings showing the diastolic gradient The uppermost tracing is an intracardiac phonocardiogram from L V

the other hand, whenever a diastolic pressure gradient across the mitral valve is absent or minimal, and there is a significant rise of mean pressure (4 mm Hg or more) during ventricular systole, it is apparent that some blood flowing into the atrium must come from a high blood pressure chamber, i e , the left ventricle If the pattern of the pulse in the atrium resembles that of the ventricle then no other possibility can be accepted

The study of pressure patterns revealed the following typical configurations

(a) Four cases of *pure or severe predominant mitral stenosis* were observed There was either *a high A wave* (sinus rhythm) or no abnormality of the pattern (Fig 47)

(b) Five cases of *pure mitral insufficiency* were studied There was a triangular *late systolic wave (I wave or insufficiency wave)* which only occasionally fused with the following (early diastolic) V wave (Fig 48) The authors call this wave insufficiency wave ' (hence the symbol I) because the highest peak appears before or at the second heart sound

(c) Fifteen cases of *mitral stenosis plus insufficiency* were observed They usually showed an entirely new pattern which consisted of a *plateau like wave* during ventricular systole Three variations of this pattern were found

(1) The A V notch was followed by a drop which however failed to reach diastolic level This was called an intermediate pattern and interpreted as evidence of *minimal insufficiency* in the presence of severe stenosis (Fig 49)

(2) There was a *large A wave* After this the pressure failed to return to diastolic level but stayed high then dropped without reaching the diastolic level during systole This also was considered evidence of *moderate regurgitation in the presence of stenosis*

(3) With either sinus rhythm or atrial fibrillation there was *a plateau like wave* during ventricular systole (Fig 50) This showed either a flat top or an oblique rising line The end of the plateau was before at or after the second sound This pattern was interpreted as evidence of *slight to moderate insufficiency in the presence of moderate to severe stenosis*

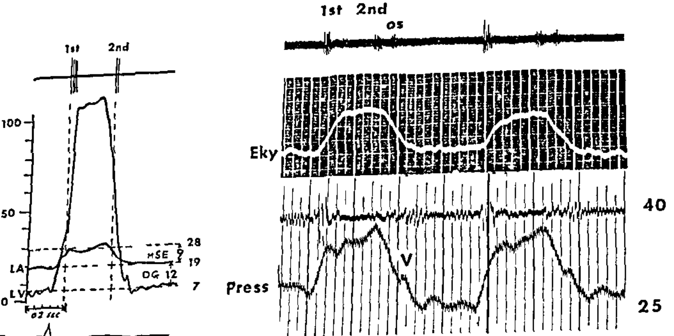


Fig 50—Cases of mitral stenosis and insufficiency
(A) Superimposed left ventricular and left atrial pressure tracings Systolic plateau in the LA pressure curve
(B) From above phonocardiogram
electrokymogram of left atrium (systolic plateau)
phonocardiogram
pressure pulse of left atrium (systolic plateau)
(From Am J Card 1958)

The drop of pressure at the end of the plateau started before the second sound when left atrial pressure was very high, at the second sound in the average cases, and at the time of the V wave in the few cases having only a minimal stenosis Thus, the end of the plateau behaved either like an I or an I V wave

The importance of the left atrial pressure is revealed by cases with severe ventricular arrhythmia due to atrial fibrillation In cycles preceded by a long diastole, where the left atrium empties well, the regurgitant pattern assumes a triangular aspect On the contrary, in cycles preceded by a short diastole, where the left atrial pressure is higher (engorgement of the atrium), a better transmission of the left ventricular pressure occurs and a squarish plateau can be observed

It is surprising that *pure insufficiency* should give a late systolic, triangular wave while *insufficiency plus stenosis* should give a plateau like

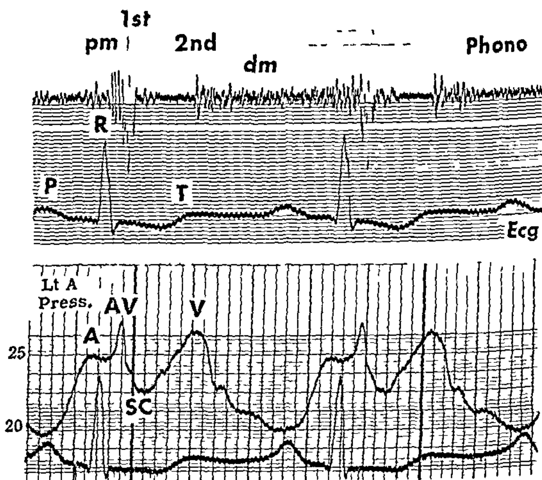


Fig 49—Phonocardiogram and ECG (above) superimposed over a left atrial pressure tracing in a case of pure mitral stenosis
A = presytolic atrial wave
AV = wave caused by mitral valve swing
V = early diastolic wave
S C = systolic collapse

tricular failure and is more common with atrial fibrillation than without it (a normal A V sequence of contraction insures a better closure of the mitral valve)

An abnormal M complex pattern can be found in cases with impaired ventricular diastole due to constrictive pericarditis, amyloidosis, fibro elastosis or fibrosis of the left ventricle (Fig 53)

Abnormal Left Ventricular Patterns

Three patients of *aortic insufficiency* were studied Two had normal left ventricular diastolic pressure while one had a moderate elevation of this

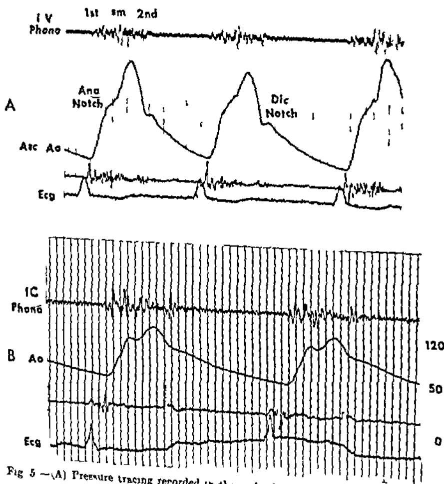


Fig 5 —(A) Pressure tracing recorded in the arch of the aorta in a patient with aortic stenosis Note the anacrotic and dicrotic notches and the late systolic peak Ill defined diamond shaped systolic murmur in the aortic phonocardiogram
(B) The aortic pressure tracing shows a double peak with the last peak higher than the first Patient with aortic insufficiency (same patient as in 51 A) Irregular systolic murmur and early diastolic vibrations are present in the intra aortic phonocardiogram

pattern One possible explanation is that mitral stenosis, with its high atrial diastolic pressure, keeps the atrial walls under a state of persistent tension Then, if regurgitation occurs, a more accurate reproduction of ventricular pressure takes place in the atrium Still, it might be postulated that, during the first half of ejection, the musculature of the left ventricle tightens so much the mitral ring that it prevents regurgiation This would explain why *a typical squarish plateau is observed in cases of left ven*

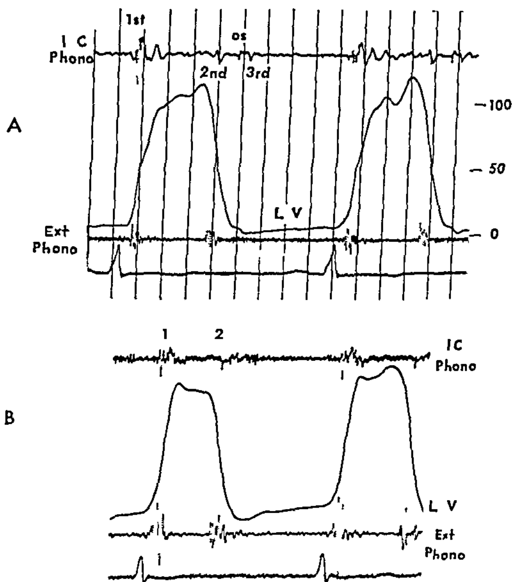


Fig 51—(A) Left ventricular strain pattern with a late systolic peak recorded in a patient with aortic insufficiency Opening sound (o s) of the mitral valve and third sound (3) are shown in the intracardiac phonocardiogram (upper tracing) (the diastolic murmur was not recorded)

(B) A late systolic peak of LV pressure curve is shown only in the second cycle The patient had atrial fibrillation and aortic insufficiency *The diastole* preceding the first cycle was shorter than that preceding the second The intracardiac phono shows a minimal early diastolic murmur

pressure The two patients, who had no evidence of left ventricular failure, had an abnormal ventricular pattern The pattern is similar to that which has been already described for the right ventricle with the name of "mechanical strain pattern," and presents a late systolic peak (Fig 51 A and B) The term "mechanical strain" does not explain the pathophysiological mechanism which is responsible for the production of this abnormal pattern Therefore, the term should be considered speculative

In a patient with *myocardial fibrosis* of unknown etiology, in whom the left ventricle had an increased resistance to filling during diastole, there was a rapid early diastolic dip immediately followed by a rapid upward swing (Fig 53) Clinically, this patient was suspected to have mitral

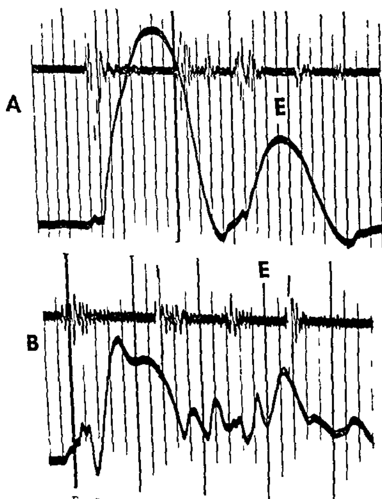


Fig 54—Ventricular premature contractions (E E)
(A) Right ventricular pressure pulses
(B) P A pulses

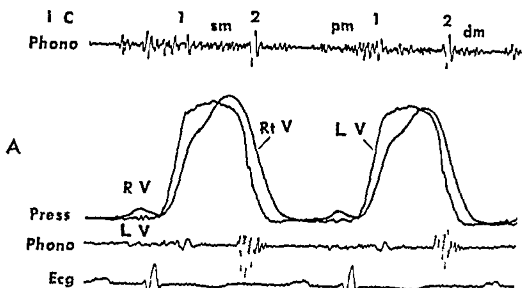


Fig 53—(A) Left and right ventricular pressure tracings (same calibration) in a patient of atrial septal defect with right to left shunt The right ventricular pressure tracing shows a late systolic peak The intracardiac phonocardiogram of the *left ventricle* reveals a presystolic murmur (PM) a systolic murmur (SMO) and an early diastolic murmur (DM) No murmur is present in mid diastole The ventricular diastolic pressures are almost identical As this indicates same pressures in the two atria it may explain why there was no diastolic murmur in that phase

(B) A pullback tracing from the left ventricle to the left atrium in a patient with myocardial fibrosis of unknown origin Early diastolic dip in the left ventricular and atrial pressure tracings The left atrial pressure pattern is M shaped

wave without irregularity in their sequence (Fig 55) *Left ventricular alternans* indicates damage or functional deterioration of the left ventricular myocardium *Right ventricular alternans* was described by Katz and associates on the basis of cardiac catheterization It was subsequently observed by us in two clinical cases and interpreted as evidence of severe deterioration of the right ventricular function

Nodal rhythm, a disturbance of the rhythm consisting of simultaneous *atrial and ventricular contractions*, may cause typical changes in the *atrial and caval patterns* (Fig 75) The rise in pressure due to atrial contraction takes place during *part* of ventricular systole and can thus be differentiated from the result of valvular incompetence which would give a plateau wave during most or all of systole

Atrial flutter Flutter waves have been described in the tracings of the right atrium right ventricle and pulmonary artery While the atrial waves are certainly due to rapid and coordinated contraction of the atrial walls those of the ventricle and of the pulmonary artery are likely to be caused by an unavoidable artifact (shaking of the catheter in the tract passing through the right atrium) We have recorded them both in the left and right atria

Atrial fibrillation Atrial fibrillation is always revealed by the absence of the A waves In certain cases, the authors have observed multiple, small waves either in the right or the left atrium These waves occurred without any regular sequence and were interpreted as caused by irregular contraction of bundles of fibers of the atrial musculature

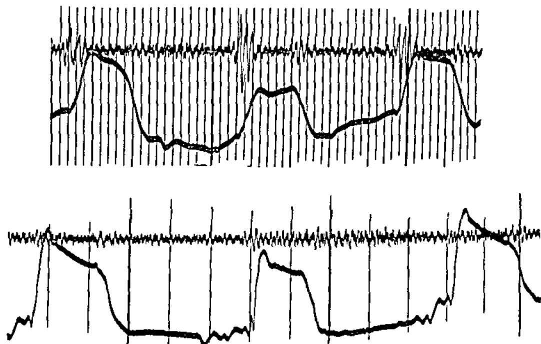


Fig 55—Alternans of the right ventricle in two cases of cor pulmonale

stenosis because of an opening snap heard by auscultation and registered by the phonocardiogram Actually, this sound was a third heart sound occurring 0 09 sec after the second sound, it was due to the early, rapid filling of the left ventricle as a result of myocardial damage and fibrosis There was no evidence of either mitral stenosis or insufficiency

Aorta

A delayed or notched upstroke of the aortic pressure tracing has been recognized as evidence of *aortic stenosis* Such a tracing was recorded in the ascending aorta of 8 patients of aortic stenosis through arterial retro grade catheterization (Fig 52) Often numerous vibrations (equivalent to a thrill and caused by turbulent flow) are recorded, and there is a slow rise of the aortic pressure tracing plus, at times, an anacrotic notch (Figs 52, 64 and 65) As already described, in cases of aortic stenosis, a marked difference in pressure exists between LV and AO This differ ence can be revealed by either a pullback maneuver or simultaneous tracings (Fig 56)

Arrhythmias Alternans

Ventricular premature contractions are revealed by *smaller waves* in the tracings of the right ventricle or pulmonary artery (Fig 54) The small wave is nearer the preceding wave and is followed by a pause which may (ventricular premature contractions) or may not (atrial or nodal premature contractions) be compensatory

Alternans is revealed by the regular sequence of a large and a small

wave without irregularity in their sequence (Fig 55) *Left* ventricular alternans indicates damage or functional deterioration of the left ventricular myocardium *Right* ventricular alternans was described by Katz and associates on the basis of cardiac catheterization It was subsequently observed by us in two clinical cases and interpreted as evidence of severe deterioration of the right ventricular function

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CHAPTER EIGHT

Abnormal Pressure Measurements and Their Interpretation

Pulmonic and Aortic Stenosis

Evidence of pulmonic or aortic stenosis has been discussed in the previous chapter Impressive results are obtained by recording *simultaneous* pressure tracings of the pulmonary artery and the right ventricle, or of the aorta and left ventricle, with the same calibration and the same zero reference line (Fig 56) As an alternative, one can record a pressure tracing while making a pullback maneuver across the pulmonic valve (pulmonic stenosis) (Fig 57 A) or the aortic valve (aortic stenosis), (Fig 57 B) A pressure tracing during a pullback across a stenotic valve guarantees the same sensitivity of pressure transducer, amplifier and recording system, and the same zero reference line

It should be kept in mind that a pullback pressure tracing across a semilunar valve cannot detect a dynamically insignificant stenosis For example, no systolic gradient of pressure between the aorta and left ventricle was found at rest in a patient with a systolic, grade III murmur over the aortic area The patient died eight months later At autopsy, the aortic leaflets were fused and calcified, forming a triangular orifice estimated to have an area of 0 9 cm² When a semilunar valvular orifice is reduced to a smaller area (0 6 cm²), ventricular systolic pressure becomes higher than that of its respective great vessel

Since the calculation of the functional valvular area of a narrowed valve requires mean systolic pressure difference the pressure measurement of mean systolic pressure of a ventricle and of its respective great vessel is important *Mean systolic pressure of an artery* is measured from the beginning of the upstroke to the dicrotic notch Likewise, *mean systolic pressure of a ventricle* is measured from the point where the pressure just exceeds that of the artery to the highest point This measurement should include several systoles in one or more respiratory cycles or in 10 consecutive beats (patients with atrial fibrillation) The pressure gradient across the aortic valve may be moderate or reach extremely high values, even 100 mm Hg or more

The square root of the mean systolic pressure gradient which is proportional to the degree of narrowing of a stenotic orifice, is termed *stenosis index* The authors usually suggest pulmonic valvotomy in

cases of isolated pulmonic stenosis when the pulmonic stenosis index is *greater than 8*

One patient a housewife who had a mean systolic pressure gradient of 80 mm Hg across the pulmonic valve and had a calculated pulmonic valvular area of 0.38 cm² refused pulmonic valvotomy However and against medical advice she had two subsequent uncomplicated pregnancies followed by normal deliveries

In aortic stenosis the authors usually advise aortic valvotomy when an adult has a stenosis index *greater than 7* with repeated episodes of left heart failure The authors would like to emphasize that clinical judgement is as important as the magnitude of the stenosis index and the calculated functional valvular areas On the other hand, clinical judgement alone is frequently inadequate if not correlated with technical data

Pulmonic and Aortic Insufficiency

We have pointed out in the previous chapter that the diagnosis of pulmonic insufficiency is very difficult because of the low diastolic pressure *existing* in this vessel even in normal subjects In severe aortic insufficiency aortic diastolic pressure is usually below 40 mm Hg, but in many cases this cannot be verified by pressure tracing

Mitral and Tricuspid Stenosis and Insufficiency

Pressure patterns of the mitral and tricuspid lesions have been discussed in the previous chapter Since mitral valve lesions occur much more often and are more important the pressure measurements of the left atrium (systole and diastole) and of the left ventricle (diastole) have been discussed in detail

The necessary requirements for a correct evaluation are (a) patient at rest, (b) evaluation during one (or possibly more) pullback maneuvers,

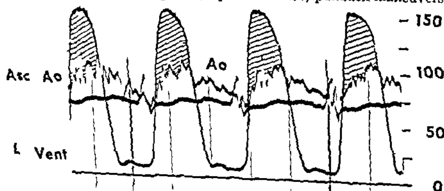


Fig 56—Left ventricular pressure tracing superimposed over aortic pressure tracing (patient of aortic stenosis) Systolic vibrations corresponding *to the* murmur occur in the aortic pressure *tracing* The systolic pressure gradient between the left ventricle and the aorta is shown by shaded area

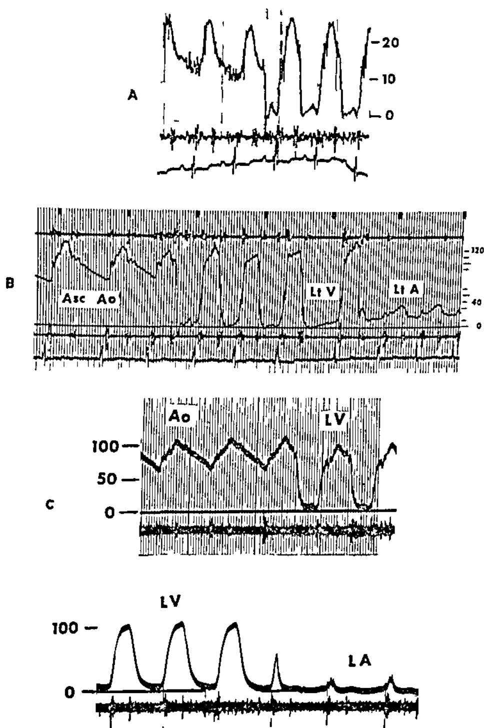


Fig 57—(A) Pullback maneuver from pulmonary artery to right ventricle In this 14 year old girl the electrocardiogram showed incomplete right bundle branch block The pressure tracing is underdamped No gradient is revealed by the maneuver

(B) Pullback maneuver from aorta to left ventricle then to left atrium Patient with mitral stenosis and insufficiency Diastolic gradient (LV to LA) and systolic elevation (LA)

(C) Pullback maneuver from aorta to left ventricle (upper tracing) and from the left ventricle to left atrium (below) in the same normal subject Slight overdamping of lower tracing

thus obtaining the same conditions and degree of amplification, (c) measurement of the diastolic pressure both in the atrium and ventricle at mid diastole (if there is atrial fibrillation) or just prior to the atrial contraction (in cases with sinus rhythm) and (d) an average of at least 10 cycles for the atrium and the ventricle during normal respiration (Fig 58)

In cases with sinus rhythm *the highest point prior to the* presystolic A wave (0 14 sec before the Q wave of the ECG) should be accepted as the *reference level* (Fig 47 C) In cases with atrial fibrillation while diastolic atrial pressure may be high during short diastoles long diastoles often allow the pressure to drop gradually to near normal levels (Fig 59) For this reason the pressure measured at about one half of diastole (average of 10 similar cycles), should be accepted as the actual diastolic level of atrial pressure

Dynamically significant mitral stenosis should be admitted when *a gradient of more than 5 mm Hg* is found between the diastolic pressure of the left ventricle and that of the left atrium (Fig 58) Even this limit should be considered as conservative because a perfectly normal mitral valve shows *no* gradient (Fig 34, 57 C) However it should be kept in mind that any existing gradient would be increased by greater venous return and usually exercise increases the gradient For this reason an *exercise test* should be made in doubtful cases The pressure gradient across the mitral valve can vary from 5 to 30 mm Hg Pulmonary edema develops when the pulmonary artery wedge pressure is above 32 mm Hg (Fleury) for any length of time However three of our patients had mean left atrial pressure of 32 mm Hg or higher without clinical evidence of pulmonary edema One of them had a mean left atrial pressure of 38 mm Hg This observation is interesting and deserves further investigation

Mitral Regurgitation With or Without Stenosis

A *high systolic insufficiency wave* (Fig 59) (I wave or I V wave) in a direct left atrial pressure measurement (Fig 59) or in a pulmonary artery wedge pressure tracing (indirect left atrial pressure) has been described as the result of mitral regurgitation However any proportion between the height of the systolic I waves or I V waves and the severity of mitral regurgitation has been denied by others Several methods of study of the left atrial pressure pulse have been described (Owen and Wood, Morrow, et al Facquet et al) in order to distinguish the pattern of significant mitral insufficiency from that of mitral stenosis However due to a non linear response of the pressure to changes whenever there is an increase of left atrial volume an exact evaluation of the severity of mitral regurgitation in the presence of predominant stenosis is still impossible

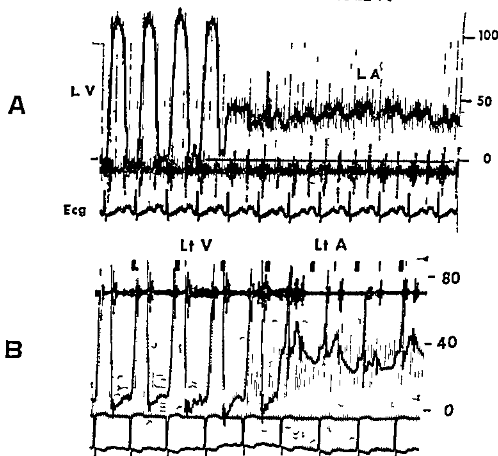


Fig 58—(A) Pullback maneuver from left ventricle to left atrium in a patient with severe pure mitral stenosis
(B) Pullback maneuver from left ventricle to left atrium in a patient with severe mitral stenosis and minimal insufficiency The intracardiac ECG (upper ECG) ($V_{LV} \rightarrow V_{LA}$) shows normal Qs complex but an abnormal slightly elevated ST segment and a positive T wave The lowest tracing is an ECG in lead 2

Left atrial mean pressure should be measured during ventricular systole by planimetric integration of the segment between the A V notch and the V wave (for 10 consecutive systoles in one or more respiratory cycles) The heart sounds recorded in a phonocardiogram are excellent for timing the opening of the mitral valve Diastolic pressure of the left atrium and ventricle has been described previously A mean elevation of the left atrial pressure during ventricular systole above 3 mm Hg over the atrial diastolic pressure indicates mitral insufficiency The square root of this elevation is termed *mitral insufficiency index*

In the presence of severe mitral stenosis with a stenosis index above 3 16, the insufficiency index (square root of the systolic elevation of the mean left atrial pressure above atrial diastolic pressure) cannot be correlated with the amount of regurgitation because the atrial wall is continuously kept under great tension On the other hand, a slight increase

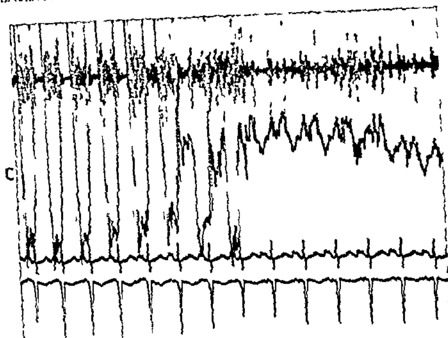


Fig 58 (*continued*)

(C) Pullback maneuver from left ventricle to left atrium in patient with severe mitral stenosis and minimal insufficiency The intracardiac phonocardiogram (upper tracing) reveals a diastolic murmur in the left ventricle which almost disappears in the left atrium Lowermost tracing = V lead from cardiac chambers Above it = lead I The peak of V LA coincides with the 2nd peak of the notched P wave in lead I

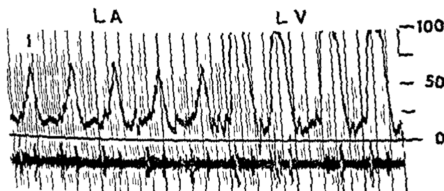


Fig 59—Push forward maneuver from left atrium to left ventricle in a patient with pure mitral insufficiency Note the high V wave coinciding with the second sound

of mean left atrial pressure during ventricular systole usually indicates a significant mitral regurgitation if the stenosis index is below 3 16

The left atrial pressure pulses analyzed by the method of Owen and Wood modified by Morrow et al is useful in order to select patients with pure or predominant stenosis According to Owen and Wood P_1 =

pressure of I-V waves, P_2 = Y descent, T_2 to T_1 = time in seconds from P_1 to P_2, Ry ratio $= P_1 - P_2/T_2 - T_1$ mm Hg/sec Index of Owen and Wood = Ry/P_1 ratio Index of Morrow et al = Ry/La mean However, in the presence of severe mitral stenosis, the value of this index for detection of significant regurgitation is open to question The height of the triangular wave of insufficiency may reach even 70 mm Hg This pressure would be incompatible with life if sustained Actually in our cases, the *mean* atrial pressure was never higher than 30 mm Hg

CHAPTER NINE

Intracardiac Phonocardiography in Mitral and Aortic Valve Lesions

In our Laboratory 17 clinical cases were studied. They were submitted to left heart catheterization according to the method of Bjoerk with the modifications of Kent et al and Fisher et al. The patients were in a prone position over a fluoroscopic table and a needle was introduced into the left atrium from the right of the spine. Pressure tracings were recorded with a *Statham* P 23 D strain gauge. The recording apparatus was a 6 channel universal cardiograph with cathode ray oscilloscopes built by *Electronics for Medicine*.

Intracardiac phonocardiograms were recorded according to previous description (Chapter five) by connecting the strain gauge channel by means of a short cable to the phono channel. The medium and high frequency vibration of the gauge was differentiated, amplified and filtered, then recorded by the phono channel. This system allows one to register the vibrations of the blood within the various chambers without introducing any additional device into the heart. Registration can be made from the needle (left atrium) or from a thin polyethylene catheter (left atrium, left ventricle, aorta). The polyethylene catheter is used for left ventricular catheterization and for retrograde aortic catheterization (following puncture of the brachial artery with a 1½ inch long 18 gauge thin walled needle).

The pressure tracings were recorded at that degree of amplification which was most suitable for obtaining a satisfactory tracing. The intracardiac phonocardiogram was recorded in the band 60 to 110, 60 to 250 or 60 to 500. The band 30 to 110 was also used. It should be noted that the degree of attenuation of the filters is gradual,* so that higher and lower pitched vibrations were still recorded though minimized.

Severe Mitral Stenosis (mitral insufficiency, minimal or absent)

Tracings recorded in the left atrium revealed *no* systolic murmur in 2 cases and a few low frequency systolic vibrations in decrescendo in 1 case. A *fourth* case had an opening snap of the mitral valve. Tracings recorded in the left ventricle of 1 case (Fig. 60) revealed an opening snap of the mitral valve, the largest vibration of which followed the aortic component of the second sound by 0.05 sec. Following this snap, a diastolic rumble with presystolic accentuation was clearly visible. The largest vibration of the first sound followed the Q wave by 0.075 to 0.080 sec. In another case the left ventricular tracing revealed the

* The filters decreased the signals at a rate of 12 decibels per octave respectively above and below the selected frequencies.

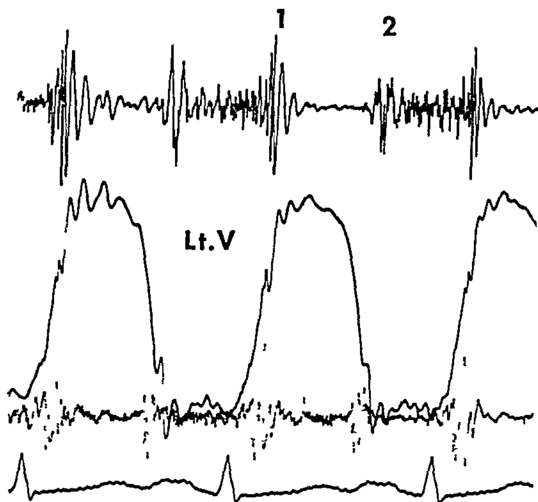


Fig 60—Intracardiac phonocardiogram of the left ventricle in a patient with mitral stenosis In the short diastole there is a diastolic murmur with presystolic accentuation

following data in the band 15/250, a prolonged opening snap and a third sound and, in the bands 30/250 and 60/500 (slight pullback), a prolonged all diastolic rumble

Mitral Stenosis and Insufficiency

The *left ventricular tracings* revealed abnormalities which were similar to those described for pure stenosis One case has atrial fibrillation and showed a gradually fading diastolic murmur whenever diastole was prolonged In the cycle which followed a short diastole, the diastolic murmur was larger and more prolonged

The *left atrial tracings* revealed an opening snap of the mitral valve in 4 cases out of 7 They also revealed the vibrations of a systolic murmur in 5 out of 7 cases This murmur was larger after a longer diastole and at times, absent after a short one (Figs 61 and 62) Diastolic vibrations were recorded in 1 case

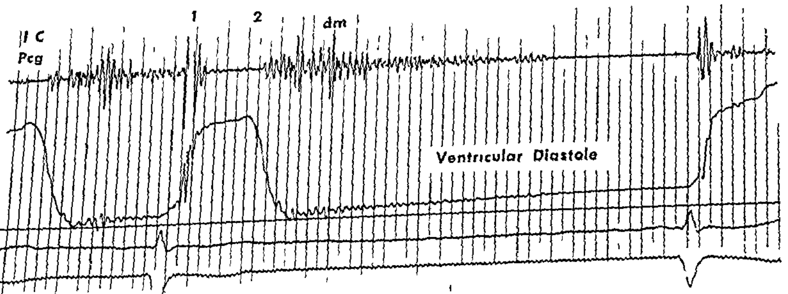


Fig 61—Intracardiac phonocardiogram of the left ventricle (IC Pcg) in a patient with moderate mitral stenosis and insufficiency atrial fibrillation and long diastoles Note the amplitude of the diastolic murmur during early and mid diastole and the absence of any diastolic murmur in late diastole (1 sec after the second sound)

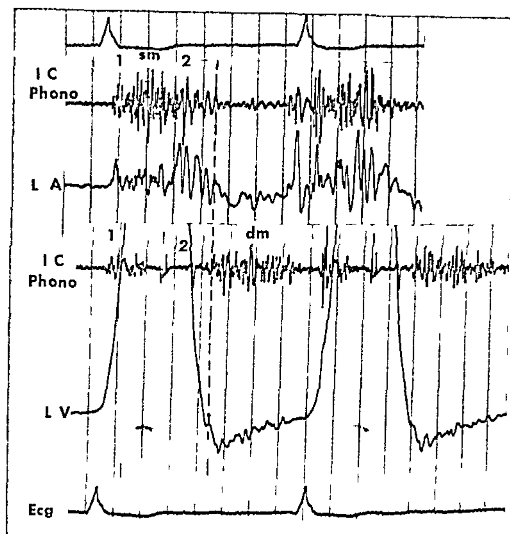


Fig 62—Superimposed left atrial (*upper*) and left ventricular (*lower*) intracardiac phonocardiograms in a patient with mitral stenosis and insufficiency The systolic murmur is best recorded in the left atrium (LA) and lasts until early diastole while the diastolic murmur begins at this moment and is recorded only in the left ventricle The atrial pressure tracing is somewhat underdamped

Mitral Insufficiency (mitral stenosis minimal or absent)

Left ventricle The ventricle was not entered in 1 case In the other 2 cases, a systolic murmur was recorded In one, a short and small diastolic rumble was recorded

Left atrium The left atrium of these 3 cases (as well as that of 7 cases with mitral insufficiency and stenosis) revealed the vibrations of a *systolic murmur* This was usually shorter and of smaller amplitude after a short diastole It was better recorded through the catheter than through the needle and became particularly large when the tip of the catheter approached the mitral valve (Fig 63)

In one case of combined valvular lesions, in which the phonocardio

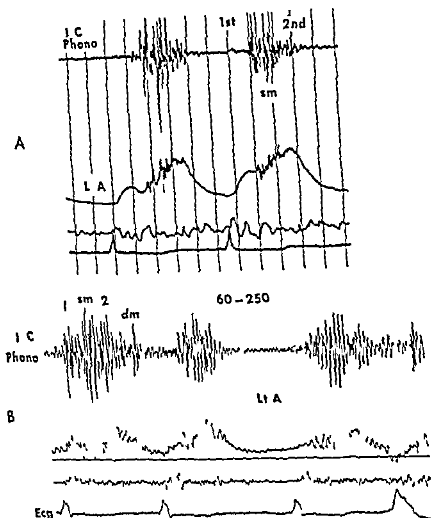


Fig 63—(A) Left atrial intracardiac phonocardiogram in a patient with pure mitral insufficiency It shows a diamond shaped systolic murmur followed by a low amplitude diastolic murmur Note the amplitude of the systolic murmur which becomes smaller after a shorter diastole (second complex) The systolic murmur is also revealed by the atrial pressure tracing (thrill felt by the operator while holding the needle) The pressure tracing is somewhat under damped

(B) Left atrial intracardiac phonocardiogram in patient with pure mitral insufficiency The tracing shows a systolic murmur which begins after the first third of systole (strain gauge directly connected to the needle) The pressure tracing reveals the thrill

grams revealed an apical systolic murmur and the left atrial pressure tracing revealed only a minimal systolic elevation, the *i c phono of the* left atrium showed large vibrations in late systole coinciding with a *thrill* in the pressure tracing It was interpreted as due to minimal insufficiency

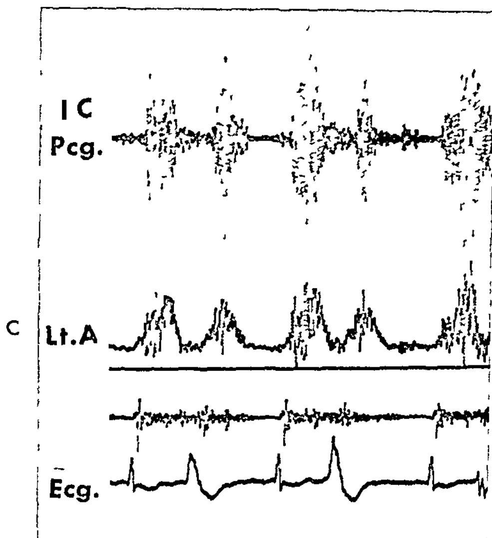


Fig 63 (*continued*)

(C) Same tracing as in (B) at lower film speed Larger amplitude of the systolic murmur after a longer diastole

Aortic Stenosis and Insufficiency

The tracings recorded in *the ascending aorta* chiefly revealed the systolic murmur (Figs 64 and 65) A diastolic murmur was apparent in cases with severe insufficiency while it was *nonexistent in the others* (Fig 65) The ventricular and atrial tracings revealed the data pertinent to mitral lesions if they were present with the exception of a functional diastolic rumble in the left ventricle recorded in 1 case A patient with aortic stenosis and insufficiency and *no* gradient across the mitral valve presented a large but late diastolic murmur within the left ventricle This was interpreted as due to eddies in the left ventricular chamber (relative stenosis—Austin Flint murmur) (Fig 66)

In the 17 studied cases the hemodynamic diagnosis was based on the details of the pressure pattern of the left atrium, as well as on the study

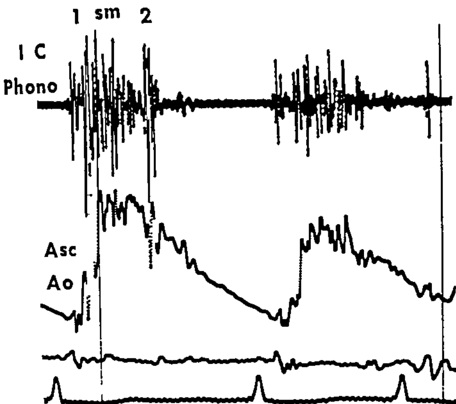


Fig 64—Intravascular sound tracing of the ascending aorta in a patient with aortic stenosis The systolic murmur (SM) is diamond shaped The pressure tracing reveals a marked systolic thrill

of pressure gradients The external phonocardiogram and the various clinical roentgenological and electrocardiographic data were often in agreement with those of catheterization while in some cases, there was some discrepancy In certain cases the diagnosis of mitral insufficiency and stenosis was entertained The presence of a large systolic murmur *in the left atrium* confirmed and increased the value of catheterization data (systolic elevation of pressure) If, on the other hand, the pressure pattern was normal and a systolic murmur was found only in the vicinity of the mitral valve it was accepted as evidence of minimal (dynamically insignificant) mitral insufficiency If the systolic murmur was present *only in the ventricle* then a different mechanism was postulated (functional mechanism) If it was *only or mainly in the aorta* aortic stenosis (absolute or relative) was recognized as the cause

The finding of a large diastolic rumble *in the left ventricle* was usually evidence of mitral stenosis However, an exceptional case where the pressure gradient across the mitral valve was normal was observed In this case as there was aortic insufficiency the murmur was considered

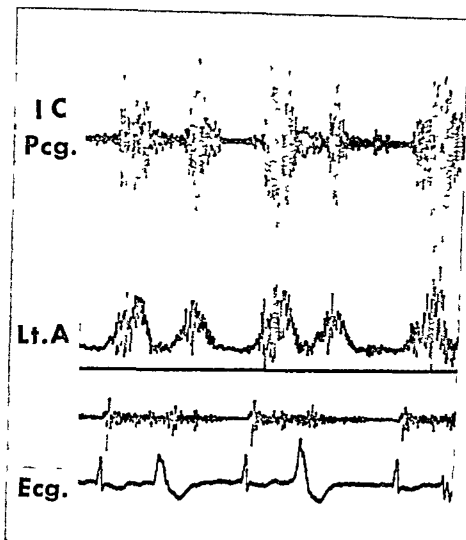


Fig 63 (*continued*)
(C) Same tracing as in (B) at lower film speed Larger amplitude of the systolic murmur after a longer diastole

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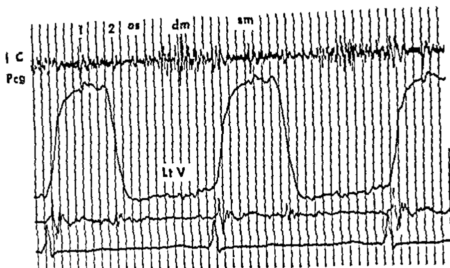


Fig 66—IC phono of the left ventricle in a patient with rheumatic aortic insufficiency The opening sound of the mitral valve (O S) occurs 0 12 sec after the second sound The diastolic murmur begins shortly after the O S but has the greatest intensity at mid diastole and is diamond shaped (Austin Flint murmur)

contraction (atrium) and (c) at the time of rapid filling (ventricle) These are physiological vibrations and do not indicate lesion of the mitral valve

In 3 cases with *ventricular arrhythmia* it was noted that short diastole was followed by a smaller systolic murmur in the atrium and a larger diastolic murmur in the ventricle (Fig 60 B) This indicates that the murmurs were flow murmurs and were affected by the amount of blood existing in the chamber from which the motion of the blood started causing the murmur (smaller systolic murmur because of lesser ventricular filling and larger diastolic murmur because of engorgement of the atrium) This phenomenon was particularly evident in regard to systolic murmur Whenever a very early contraction caused an extreme abbreviation of diastole the systolic murmur disappeared

Intracardiac phonocardiography is barely beginning to *yield results* It is too early for a complete evaluation of the possible diagnostic advantages which this method may have in comparison with others, since only an extensive study and a continuous comparison with other existing methods may lead to definite conclusions

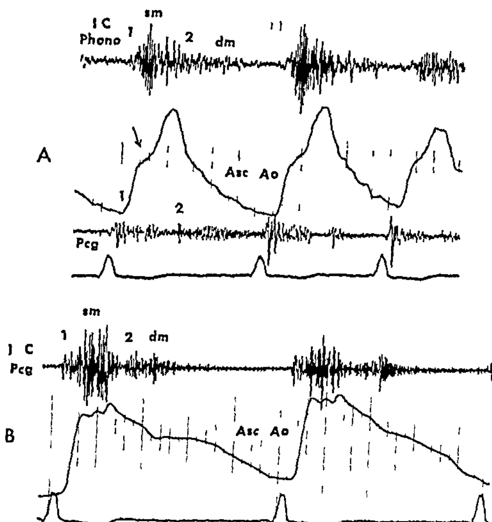


Fig 65—(A) Intravascular sound tracing of the ascending aorta in a patient with aortic stenosis and insufficiency The largest amplitude of the diamond shaped systolic murmur coincides with the anacrotic notch of the pressure tracing (arrow) This is because at that time a more marked pressure gradient develops between LV and AO The tracing shows also the early diastolic murmur

(B) IC phono of the ascending aorta in a patient with aortic stenosis and insufficiency Systolic and diastolic murmurs are shown

an Austin Flint murmur However, in other cases, the possibility of a minimal, dynamically insignificant stenosis should be considered

Timing of the heart sounds and their valvular components was found accurate The vibrations of *the first sound* within the left ventricle reflect the closure of the mitral valve, and then the opening of the aortic valve The vibrations of *the second sound* within the left ventricle reflect the closure of the aortic valve An *opening snap* of the mitral was recorded within the left ventricle (and occasionally also within the left atrium) in patients with lesions of the mitral valve On the other hand smaller vibrations may be observed (a) at the time of opening of the mitral valve (opening sound) (atrium or ventricle), (b) at the time of atrial

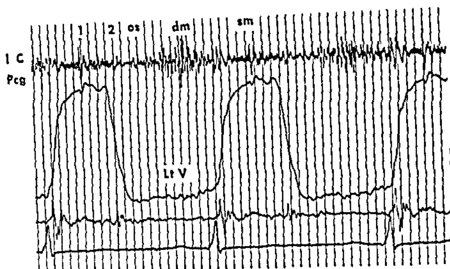


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CHAPTER TEN

Intracardiac Phonocardiograms in Congenital Heart Disease

Patent Ductus Arteriosus

An intra pulmonary artery sound tracing (Fig 67 A) and an intra right ventricle phonocardiogram (Fig 67 B) were recorded in a 20 year old male with a patent ductus arteriosus and an estimated pulmonary flow of 16 liters per miute Clinical auscultation revealed a grade II soft systolic murmur, and a grade III high pitched blowing diastolic murmur over the second left interspace and the third rib, 2 cm at the left of the sternal border An external phonocardiogram confirmed the findings of auscultation

The intrapulmonary artery sound tracing revealed a decrescendo systolic murmur with minimal amplitude before the second sound, the sound was fused with a decrescendo, all diastolic murmur of larger amplitude The amplitude of the systolic murmur was variable from cycle to cycle We cannot explain why the murmur in this case was not continuous The intra right ventricular phonocardiograms revealed no significant systolic vibrations However, an early diastolic murmur in decrescendo was recorded This murmur may be due to a transmission of the diastolic murmur from the pulmonary artery or to a minimal pulmonic insufficiency

Atrial Septal Defect

Intracardiac phonocardiograms were recorded in 6 cases The tracings of 2 patients with left to right shunt are shown in Fig 68 A–F The systolic murmur had its greatest magnitude within the pulmonary artery and was diamond shaped in both cases (Fig 68 A and D) No systolic murmur was recorded in the right atrium of the first case (Fig 68 C) In both cases, murmurs of irregular configuration or in decrescendo were recorded in the right ventricle and atrium (Fig 68 B, E and F) The second sound was delayed in both cases particularly case 1 (Fig 68 A and D) The diastolic murmur was recorded best in the right ventricle rather than in the right atrium Its beginning occurred 0 12 sec after the beginning of the second sound (Fig 58 B and E) Since the diastolic murmur is similar to those caused by mitral or tricuspid stenosis and there was no diastolic pressure gradient across the tricuspid valve while there was a large flow, the authors considered a 'relative tricuspid

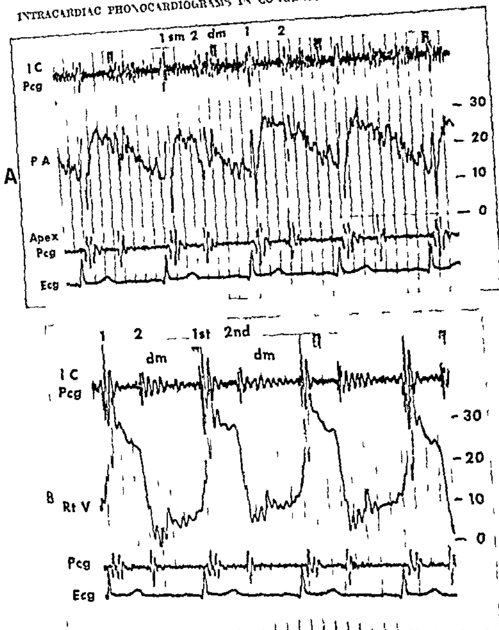


Fig 67—(A) Intravascular sound tracing of pulmonary artery (IC Pcg) in a case with patent ductus arteriosus This patient had only a minimal murmur in the external tracing the shunt was revealed by catheterization The pressure tracing (P A) is somewhat underdamped but reveals large oscillations (thrill) The sound tracing from the artery shows a systolic and a diastolic murmur
(B) Intracardiac sound tracing from right ventricle of same patient as (A) Only an early diastolic murmur is present

stenosis A presystolic murmur or a fourth heart sound is usually recorded within the various chambers of these cases (Fig 68)

The documents of one patient with right to left shunt are presented in Figs 68 G and 53 A In the left atrium and the left ventricle a presys

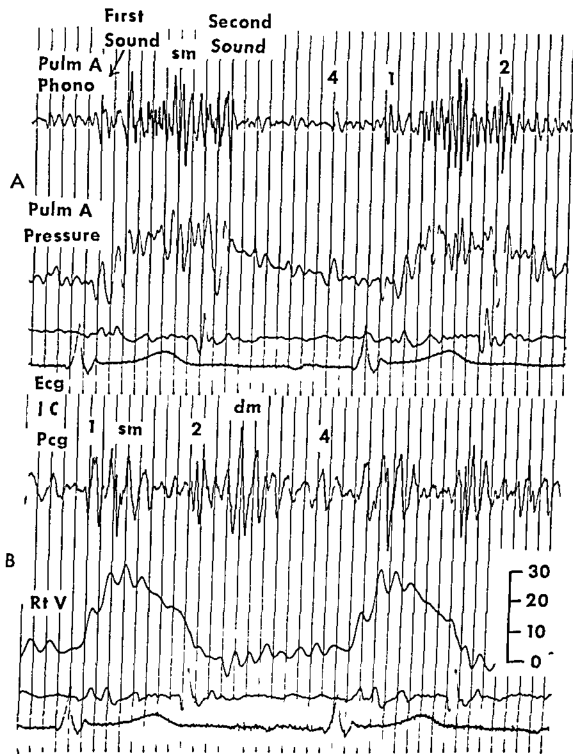


Fig 68—Cases of atrial septal defect with left to right shunt

(A) Case 1 Intra pulmonary artery sound tracing There is a diamond shaped systolic murmur and a large delayed second sound (2) The 4th sound is barely visible and is followed by minimal vibrations

(B) Case 1 Intra right ventricular phonocardiogram First sound (1) is 0 075 after the Q wave of the ECG and is followed by a decrescendo low frequency systolic murmur (sm) The second sound is delayed and has several vibrations The beginning of the decrescendo diastolic murmur (dm) follows the second sound by 0 12 sec

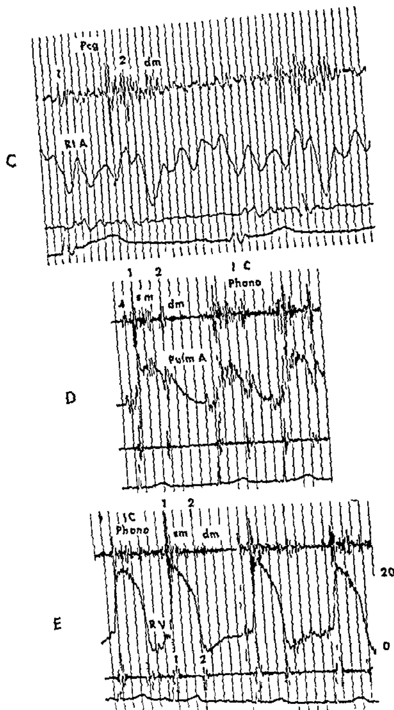


Fig 68 (*continued*)—Cases of atrial septal defect with left to right shunt

(C) Case 1 Intra right atrial phonocardiogram Minimal systolic vibrations Snapping late systolic sound Diastolic murmur greatest in early diastole but separated from the 2nd sound (delay = 0 14)

(D) Case 2 Intra pulmonary artery sound tracing Large diamond shaped systolic murmur Minimal mid diastolic murmur 4th sound

(E) Case 2 Intra right ventricular phonocardiogram Decrescendo systolic murmur Decrescendo mid diastolic murmurs 4th sound

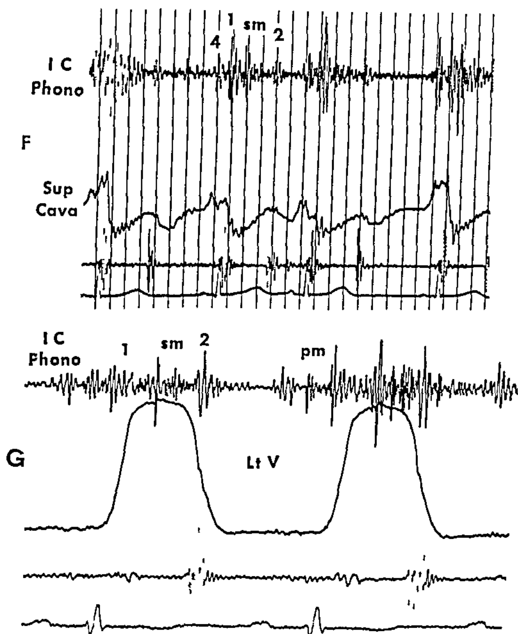


Fig 68 (*continued*)—Cases of atrial septal defect with left to right shunt
(F) Case 2 Intra superior caval sound tracing (near right atrium) 4th Sound Diamond shaped systolic murmur
(G) Case 3 Intra left ventricle phonocardiogram Presystolic murmur irregular systolic murmur early diastolic murmur

tolic murmur, a systolic murmur and an early diastolic murmur were recorded However, only a minimal systolic murmur was recorded within the right atrium and none in the right ventricle and pulmonary artery

Ventricular Septal Defect

In patients with large left to right shunt, a large systolic murmur is recorded within both the right ventricle and the pulmonary artery In a

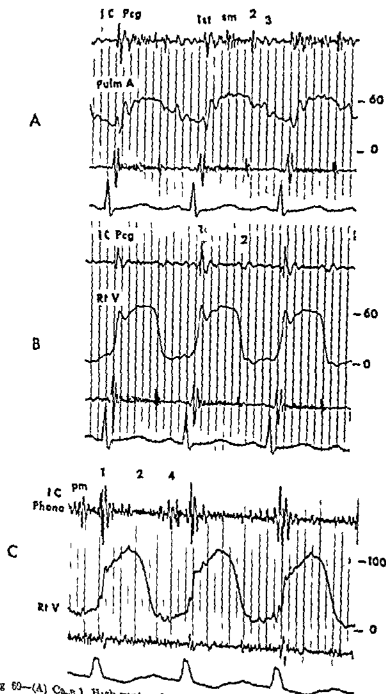


Fig 69—(A) Case 1 High ventricular septal defect Moderate left to right shunt Intra pulmonary sound tracing Irregular systolic murmurs 3rd sound
(B) Case 1 No murmur within the lower part of right ventricle
(C) Case 2 Ventricular septal defect Severe right to left shunt (and minimal left to right shunt) Presystolic murmur moderate systolic murmur

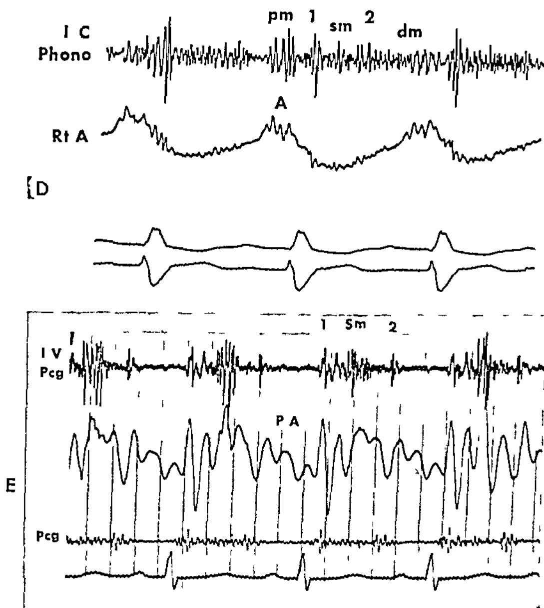


Fig 69 (*continued*)

(D) Case 2 Continuous murmur within the right atrium with presystolic accentuation
The lowest ECG is a V lead from RA

(E) Case 3 Severe pulmonic stenosis (valvular type) (same case as in Fig 39 B)
Intra pulmonic sound tracing Diamond shaped systolic murmur Delayed 2nd sound

patient with a moderate left to right shunt and right ventricular hypertension, a systolic murmur was not recorded in the RV (Fig 69 B), but was present in the pulmonary artery (Fig 69 A)

In a patient with predominant right to left shunt, a systolic murmur was recorded in the right atrium with accentuation during atrial systole (Fig 69 D) A fourth sound or presystolic murmur was also visible in the right ventricular phono tracing

Pulmonic Stenosis

A pullback maneuver from the pulmonary artery to the right ventricle with continuous i c phonocardiography is shown in Fig 39 B A diamond shaped systolic murmur was recorded in the PA and only a few low frequency vibrations were present in the right ventricle Fig 69 E shows the diamond shaped systolic murmur at a higher film speed The pulmonic second sound was delayed

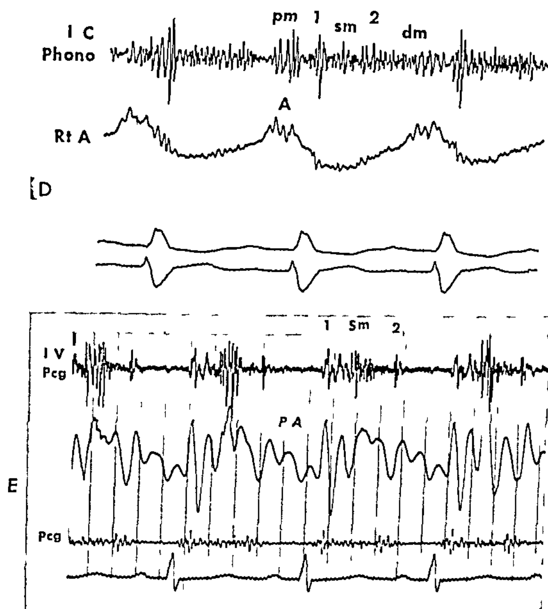


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The lowest ECG is a V lead from RA

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In a patient with predominant right to left shunt a systolic murmur was recorded in the right atrium with accentuation during atrial systole (Fig 69 D) A fourth sound or presystolic murmur was also visible in the right ventricular phono tracing

(3) In cases of *left ventricular hypertrophy or strain*—increased voltage of QRS

(4) ST and T waves alterations seem to vary according to the level of the catheter tip within the ventricle Generally the changes are in a direction opposite to those of the left precordial leads

(5) During a pullback maneuver, there was a progressive flattening of the T wave whether negative or positive However, the ventricular complex usually presented a sharp change from a ventricular to an atrial pattern both in normal and pathological cases

Electrocardiograms Recorded Within the Left Atrium

The normal patterns of atrial and ventricular complexes at various levels of the left atrium are shown in Fig 30 The most frequent pattern as recorded in the central part, is that of a *diphasic P wave* due to spreading from right to left of the wave of atrial depolarization (Fig 72 B) This followed by a QS complex and a negative T wave which are transmitted from the ventricular cavity In higher portions of the atrium, a Qr or QR pattern is consistently found the late R seems to be the expression of a late activation of basal portions of the left ventricle A diphasic (RS) ventricular activation of basal portions of the left ventricle A diphasic (RS) ventricular complex is commonly recorded in the left lateral part near the left lateral part within the left superior pulmonary vein an rSr complex is usually recorded

Cases with *atrial fibrillation* usually show only the ventricular complex, but in certain cases atrial complexes with irregular rhythm can be seen (Fig 71 B) Cases with *atrial flutter* reveal flutter waves of high voltage (Fig 71 A) similar to those which can be found in the right atrium

The *ventricular complex* at the various atrial levels becomes larger in cases with *left ventricular hypertrophy* However the voltage of this complex deflection cannot be determined in terms of absolute values (as by direct epicardial leads) because of uncertainty in regard to the distance of the tip of the catheter (transparent by X ray) from the atrial wall or the mitral valve The timing of the intrinsicoid deflection of the P waves inside the left *atrium* has been found to vary depending upon the location of the recording *electrode*, both in normal and pathological cases Our observations in the left atrium clearly indicate that the intrinsicoid deflection of the atrial complex falls in the second half of a simultaneous recorded P wave in *leads* 2 or 3, with variations in the same case of 0 02 sec according to the atrial level We have found intervals of from 0 05 to 0 09 sec between the beginning of P in lead 2 and that in the left atrial i e lead in cases of mitral stenosis Levine et al re

CHAPTER ELEVEN

Abnormal Intracardiac Electrocardiogram

In our studies,* a standardization of 0 1–0 2 N was used in each ventricle, after pullback into the atrium, the standardization was usually increased to 0 5 N At least three pullback maneuvers were obtained in every case Three different film speeds were used 25, 50 and 100 mm /sec The following findings were obtained

Left Atrial Intracardiac Tracing

Left atrial complex

(1) 7 cases with *normal sinus rhythm*—the P wave fell in the second part of the P wave in simultaneously recorded limb leads The asynchronism of the intrinsicoid deflection of the intracardiac P wave varied between 0 05 and 0 09 sec in cases with predominant mitral stenosis

(2) 6 cases of *atrial fibrillation* no evidence of co ordinated electrical activity was recorded However, in some cases, small, slightly irregular waves were recorded (impure fibrillation?)

(3) 2 cases of *atrial flutter* = F waves were recorded in both cases

Left ventricular complex The QRS presented the same characteristics as the one recorded inside the left ventricular, though usually of lower voltage In cases of *left ventricular hypertrophy*, high voltage was recorded with QS or QR patterns according to the atrial level ST and T wave alterations, parallel to those recorded in the ventricle, were noted except in one case

Left Ventricular Intracardiac Tracing

Atrial complex In cases with *sinus rhythm*, the P wave was positive and of very low voltage, due to the low amplification used In one of three cases of *pure mitral insufficiency*, the P was tall and peaked In cases of *atrial fibrillation or flutter*, no atrial activity was recorded

Ventricular complex

(1) 5 cases of isolated *right ventricular hypertrophy*—normal intracardiac tracing The T wave was always negative

(2) *Left ventricular premature beats* presented a widened QS pattern and secondary ST and T changes

* This work was done in collaboration with Dr M R Testelli

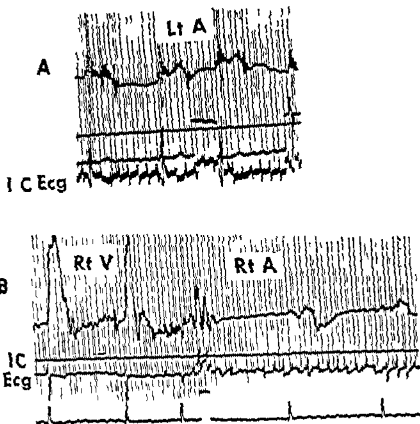


Fig 71—(A) Intracardiac electrocardiogram of the left atrium in a patient with regular atrial complexes at a rate of 360 per minute The diphasic waves are flutter waves Regular waves are barely visible in the atrial pressure tracing during diastole

(B) I C Ecg of the right cavities of the same patient

in the left atrium, great voltage was accompanied by little asynchronism, small voltage by greater asynchronism Similar findings were reported through use of direct and esophageal leads by Puech Comparing the voltage of the P waves in either atrium and that of the P waves in a standard lead, no correlation could be found in the latter, the height of the P was depending upon the axis of P in either the frontal plane (extremity leads) or the horizontal plane (precordial leads) The so called P mitrale is due to a left axis deviation of the second component (positive in 1 negative in 3) while the initial right atrial half has a normal axis As for the precordial leads mention should be made of the observation first reported by Hecht that in almost all cases of mitral stenosis a diphasic P wave is present in V_1 and that its negative part is due to the left vector of depolarization It should be accepted that the width of the P wave in the 12 lead electrocardiogram depends upon the asynchronism between the waves of activation of the two atria

We were unable to find any relationship between the height of the

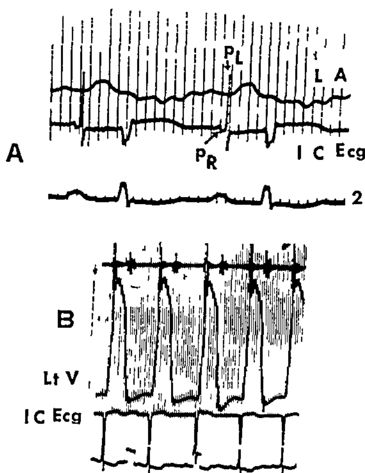


Fig 70—(A) Intracardiac electrocardiogram from the left atrium in a patient with mitral stenosis P_R = P wave of the right atrium (transmitted to the left) P_L = P wave of the left atrium (large complexes) P_L coincides with the second peak of the P wave in lead 2

(B) Intracardiac electrocardiogram of the left ventricle in a patient with left ventricular hypertrophy Note that the calibration is ½ of the standard Elevation of the ST segment and diphasic T waves in the V lead from the cavity of the left ventricle Lowest tracing = lead 2 at standard calibration

ported a delay in the left P over the peripheral P of from 0 05 to 0 07 when the catheter was in the great cardiac vein Latour and Puech, studying intracavity potentials from the left atrium in cases of congenital defects reported a delay of the left P of from 0 05 to 0 09 sec (in the same case) It follows that intracardiac and intravascular tracings, because of their nature, cannot be strictly compared to direct or semi direct leads In our cases, where catheterization of both sides was performed, no correlation was found between the P wave recorded in the right and the one recorded in the left atrium While the P wave recorded in the right atrium appears to reflect only the activity of the explored chamber, that recorded in the left is preceded by a small notching (Fig 70 A) which has been attributed to depolarization of the *right* atrium Often, in our cases, the degree of asynchronism (time of inscription of the intrinsicoid deflection) was inversely proportional to the voltage of P

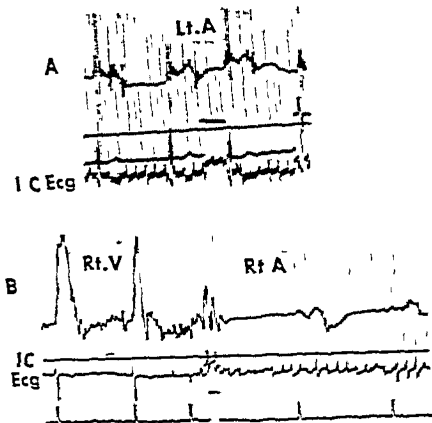


Fig. 7—A Intracardiac electrocardiogram of the left atrium in a patient with regular atrial complexes at a rate of 60 per minute. The diphasic waves are flutter waves. Regular waves are barely visible in the atrial pressure tracing during diastole.
B) I.C. Ecg. of the right cavities of the same patient.

in the left atrium great voltage was accompanied by little asynchronism, small voltage by greater asynchronism. Similar findings were reported through use of direct and esophageal leads by Puech. Comparing the voltage of the P waves in either atrium and that of the P waves in a standard lead no correlation could be found: in the latter, the height of the P wave depending upon the axis of P in either the frontal plane (extremity leads) or the horizontal plane (precordial leads). The so-called P mitrale is due to a left axis deviation of the second component (positive in 1, negative in 3) while the initial right atrial half has a normal axis. As for the precordial leads mention should be made of the observation, first reported by Hecht, that in almost all cases of mitral stenosis a diphasic P wave is present in V_1 and that its negative part is due to the left vector of depolarization. It should be accepted that the width of the P wave in the 12 lead electrocardiogram depends upon the asynchronism between the waves of activation of the two atria.

We were unable to find any relationship between the height of the

P wave recorded in the left atrium and the level of left atrial p... or that of the pulmonary capillary pressure Trounce and F... have made similar observations

The hypothesis of Reynolds that a high voltage of the left atrial P indicates a well functioning atrium in contrast to low P waves logical, even though it is still hypothetical

Electrocardiograms Recorded Within the Left Ventricle

The normal deflections consist of *a positive P wave*, followed by *QS complex* and *a negative T wave* A distinct U wave was never found, in agreement with the observations of Sodi Pallares The P wave, due to the low amplification used for intraventricular recording, is usually flat in normal subjects In one case of isolated mitral insufficiency with a systolic rise of left atrial pressure of 10 mm Hg, tall, peaked P waves were recorded in the left ventricle They had the same characteristics

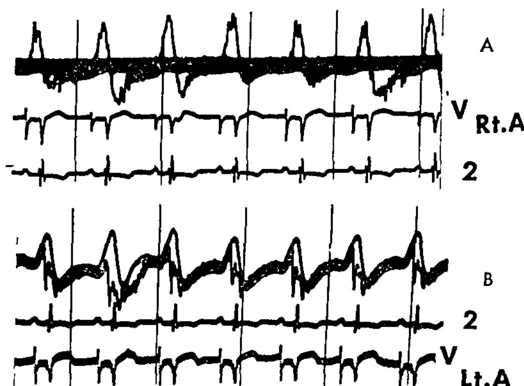


Fig 72—(A) Right atrial pressure tracing (above) *right* atrial intracardiac V lead (center) and lead 2 (below) The P wave of the i c lead coincides with the early part of P in lead 2 Atrial septal defect (case of Fig 14)

(B) Right and left atrial pressure tracings (above) ECG lead 2 (center) and intracardiac V lead of *left* atrium (below) The P wave of left atrium coincides with the second part of P in lead 2

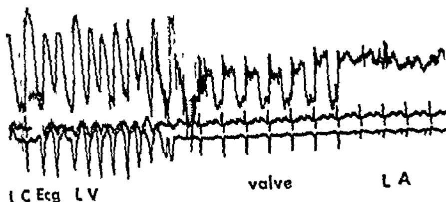


Fig 73—I C Ecg (V lead) of the left ventricle (LV) It shows runs of ventricular premature beats QS complexes in the region of the mitral valve (The catheter was moving from the atrium [ventricular systole] to the ventricle [diastole]) QR complexes in the left atrium P_{LA} is clearly visible but small on account of substandard amplification

as the P waves recorded in the left atrium the only difference was a P R interval of 0 120 sec in the ventricle and 0 185 sec in the atrium while it was 0 230 sec in lead 2

The T wave is normally inverted in the left ventricle In normal subjects during a pullback maneuver from the apex to the left atrium there is a progressive flattening of the T wave Thus a comparison between a left intraventricular unipolar lead and a left precordial lead should be made only when the catheter is in the mid portion of the left ventricle *Ventricular premature beats* elicited through stimulation of the endocardial surface by the tip of the catheter present an intraventricular pattern consisting of broad QS ST and T elevation (Fig 73) This pattern is characteristically similar to that of right ventricular premature beats when recorded within the right ventricle (Fig 75 A) However, the pattern of these premature contractions in the peripheral electrocardiogram is that of a *right bundle branch block* for premature beats arising from the *left* ventricle and a pattern of *left bundle branch block* for *right* ventricular premature beats Barker et al first revealed ventricular asynchronism occurring in extrasystoles artificially elicited through stimulation of either ventricle In 1936 Marcu followed by Loukomski and Guinodman described the pattern of premature ventricular beats in standard leads obtained by experimental stimulation of the endocardium All the following observers recognized the sensitivity of the endocardium to the mechanical stimulation by the catheter if the catheter tip presses against the endocardial surface for more than a few seconds *a monophasic wave of injury* appears which due to the

limited area of the tip of the catheter, is recorded only in the endocardial electrocardiogram (Fig 32) It is not associated with ST deviations in simultaneous precordial, standard or intracardiac leads from any other chamber Additional evidence indicating that the intracardiac injury pattern is due to a minimal, localized injury is that it disappears abruptly on withdrawal of the catheter

In cases with combined or left ventricular hypertrophy, increased *voltage of the ventricular complex was found with a positive T wave* (Fig 75 B) When a pattern of left ventricular "strain" was present in the left precordial leads, an elevation of the ST segment was recorded within the left ventricle with the catheter at the apex or in the mid portion of the chamber The same ST and T changes were also recorded within the left atrium Thus, discordance between the ST segments and T waves recorded over the precordium and those recorded within the left ventricle is found in cases with left ventricular hypertrophy, as well as in normal subjects Again, the progressive flattening of the T wave, which is seen during a pullback maneuver *from the apex to the left atrium, is not* different from that observed in normal subjects In other words, the voltage of T decreases during such a maneuver, whether it was originally positive or negative

One tracing presented special interest It was recorded in a case of mitral stenosis and insufficiency with atrial fibrillation and combined ventricular hypertrophy and strain The T waves were flat in the left ventricle while they were well visible as soon as the catheter crossed the mitral valve We have no explanation at present for this finding

Another puzzling observation consisted of the *recording of an rS pattern in the left ventricle* (Fig 74) This occurred often after a run of

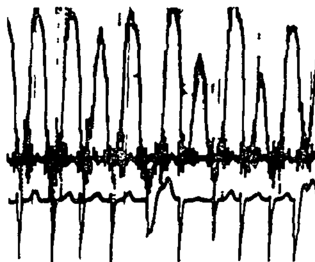


Fig 74—I C Ecg of the left ventricle VLV shows abnormal rS complexes

premature ventricular beats elicited by the catheter In two other cases, a small r was recorded during a pullback maneuver, when the catheter crossed the mitral valve Neither of the two cases fulfilled the criteria of Sodi Pallares for incomplete left bundle branch block

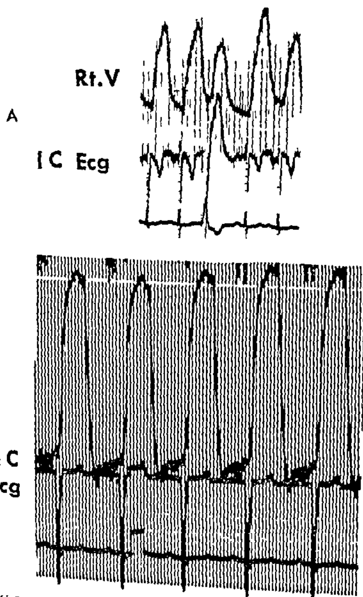


Fig 15—(A) I C Ecg of the right ventricle There is one ventricular premature beat originating in the right ventricle
(B) I C Ecg of the left ventricle in a case of left ventricular hypertrophy showing increased voltage of QRS and positive T waves

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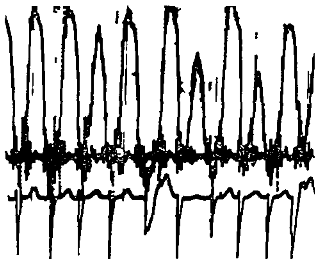


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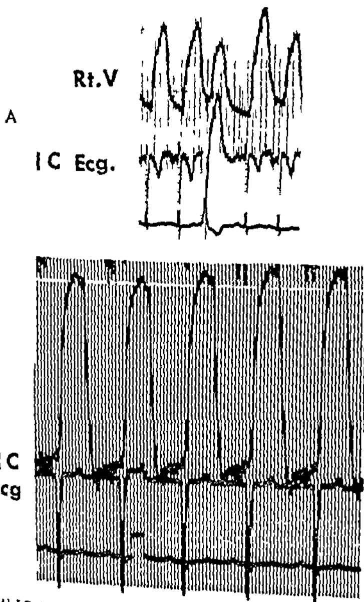


Fig 75—(A) I C Ecg of the right ventricle There is one ventricular premature beat originating in the right ventricle
(B) I C Ecg of the left ventricle in a case of left ventricular hypertrophy showing increased voltage of QRS and positive T waves

Previous reports mentioned an initial positive deflection in the left ventricle. Zimmerman reported an RS pattern with the catheter at the apex, Coelho et al. recorded an rS pattern in a normal case and in a case of left ventricular hypertrophy. Mas et al. noticed an rS pattern in the midsection of the left ventricle, but stated that this pattern was more commonly seen after numerous premature beats. Finally, Latour and Puech reported 3 instances of an rS or a qrS pattern in the left ventricle in the absence of any intraventricular conduction disturbance. The interpretation of this initial positivity is still difficult. However, following Sodi Pallares' interpretation of the spreading of the impulse in the interventricular septum, the initial r inside the upper part of the left ventricle might be explained as due to depolarization of a papillary muscle or of the highest part of the septum.

CHAPTER TWELVE

Artifacts

ARTIFACTS DUE TO FAULTY CONNECTIONS OR RECORDING

There are many factors which can produce artifacts in the patterns of the pressure pulses It is important to recognize them as early as possible during catheterization and to eliminate them immediately, as far as possible If one is not aware of some of these artifacts, the tracing of one chamber may be mistaken for that of another

The pressure recording system in cardiac catheterization consists of a radio opaque cardiac catheter connected to a three way stopcock by a Luer lock The second outlet of the stopcock is connected by sterile tubing to a bottle which is provided with a Murphy drip and contains physiologic solution The third outlet is connected either to a nondistensible lead tube or to a tube made of plastic (or a special rubber) the other end of which is connected to the transducer of either a Sanborn manometer a strain gage or a Hamilton manometer If a Hamilton recording system is used the pressure tracing can be recorded by a photographic device The electric output of the Sanborn manometer or of the strain gauge is fed into an amplifier and is recorded by a galvanometer through either a direct writing or a photographic system

From the tip of the catheter to the three way stopcock, the tubing and the chamber containing the pressure transducer should be *completely filled* with sterile 5 per cent glucose solution or a physiologic saline solution Because of the physical compressibility of gas any minute air bubble would alter the pressure tracing to some extent *This is one of the most frequent artifacts which may occur in the recording system*

If the air bubble is large, there may be no evidence of pressure variations in the tracings If the air bubble is small there will be deflections but the appearance of the pressure tracing will be changed Not only is the timing of curves delayed and the actual pressure level reduced, but the pattern of tracing often becomes indistinct and unusually smooth

When the connections between catheter, 3 way stopcock nondistensible tubing and strain gauge or manometer are not properly secured leakage tends to occur particularly if the manipulation of the catheter is carried out in a dark room and the leak is not discovered *Loose connections with slow dripping leakage in the system reduce the pressure* while only slight changes in pattern are observed They are usually recognized without difficulty while the pressures are recorded Whenever an unusually

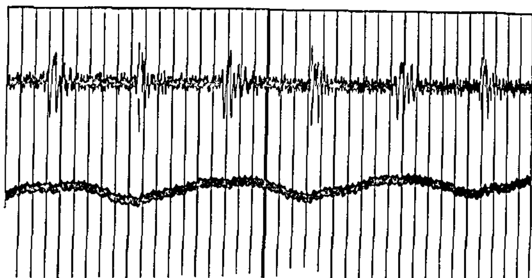


Fig 76—Small pulsations of right atrium due to initial formation of clot in the catheter

low pressure is recorded and a smooth pattern observed, the connection and the system should be checked at once

Inadequate balancing of the electromanometer due to haste (a "warming up" period of at least one hour is necessary), improper initial balancing, or subsequent imbalance result not only in improper level of pressure but also in abnormal patterns which may vary from inadequate to bizarre The same applies to *a strain gauge system* if the transducer is faulty

Improper calibration of the galvanometer system (overdamping, overshooting) in both the photographic and the direct writing systems may cause incorrect transcription of patterns

Improper application of the writing arm, too high a temperature in the writing system, or a *worn out pen* may also cause faulty tracings

All these artifacts can be avoided by checking the electrocardiograph before preparing for catheterization, following the customary rules for checking such apparatus

ARTIFACTS DUE TO BLOOD CLOTS

In order to prevent fibrin clot formation in the lumen of the catheter, it is necessary to use a continuous drip of heparinized (1000 units per liter) 5 per cent glucose solution in water at a rate of about from 40 to 60 drops per minute except when blood samples are being collected or pressure measurements recorded The catheter should be washed with 2 cc of sterile heparinized physiologic saline solution after collection of each blood sample If pressure measurements are carried out for more than a minute or two, such as during a pull back tracing, *a blood clot tends to form near the orifice of the catheter tip*, especially when a small (No 5) catheter is used If it is difficult to obtain a blood sample while the catheter tip is in the pulmonary artery or the right atrium, and it is

still possible to force washed solution fluid under pressure into the catheter, it is likely *that a small fibrin clot has formed at the tip of the catheter, narrowing the opening with a valve like effect*

After recording a long pressure tracing, it is common to observe that the graph becomes unusually smooth and the main waves become indistinct or disappear (Fig 76) In such case a small clot has usually formed One can still attempt to obtain a blood sample through gradual suction If, however this is not possible *it is unwise to force fluid* into the catheter because this might detach a clot from the wall of the latter and cause an embolism Instead the catheter should be withdrawn and a new catheter introduced into the vein (unless the essential data have been already obtained)

ARTIFACTS DUE TO MOVEMENT OF THE CATHETER

It should be kept in mind that the heart is continuously beating and that the tip of the catheter moves in conjunction with the cardiac action If this movement is prevented rhythmic occlusion of the tip is far more common The closing and opening of the cardiac valves the backward and forward movements of the catheter and the vibrations of the valves septum or arterial walls whenever murmurs are produced (Fig 42)—all can be the cause of multiple artifacts

EXAMPLES

The three tracings of Fig 77 were obtained from the same patient while the catheter was in the right pulmonary artery (A) the main pulmonary artery (B) and the right ventricle (C) In Fig 77 C the right ventricular pressure is 25/0/4 5 mm Hg (systolic/early diastolic/end diastolic) The sharp peak may be due to a slight damping of the sensitive manometer In Fig 77 B the systolic peak of the main pulmonary artery nearly reaches the same level as that of the systolic pressure of the right ventricle (23 mm Hg) indicating the normal size of the pulmonic valve There is a prominent rebound after the end of the T wave

In Fig 77 A the first notch is probably coincident with the first heart sound the second with the second heart sound both are probably of valvular origin The systolic deflection occurring in early systole is probably due to movement of the catheter and also to impact of the catheter tip against the pulmonary arterial wall

It has been stated that this systolic deflection might be due to a kind of siphon phenomenon due to the velocity of blood flow However, the absence of this deflection in Figs 77 B and C stands against this suggestion, at least in this case It is obvious that the highest pressure in Fig 77 A (17 mm Hg) is the early diastolic rebound (dicrotic wave) and that the systolic pressure of the pulmonary artery cannot be correctly measured Should one record only the deformed pattern of Fig

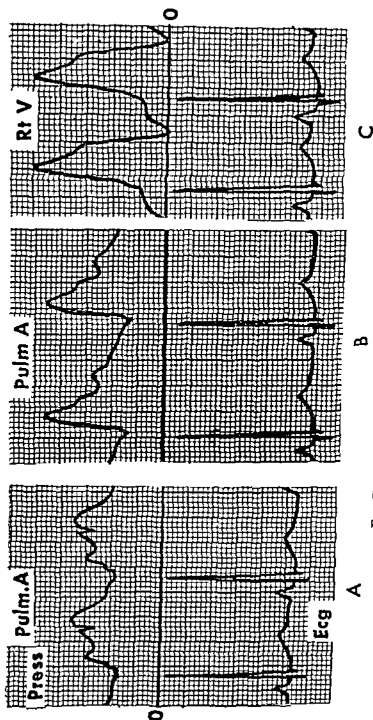


Fig 77—Artifacts in a tracing of pulmonary artery
(A) Right PA
(B) Main PA
(C) Right ventricle

77 A, one would consider the possibility of mild pulmonic stenosis on account of a gradient (artifact) between the systolic pressure of the right ventricle and the highest level of the pulse of the pulmonary artery *The use of a catheter with lateral holes near the tip would prevent the occurrence of such an artifact* This is very important because, provided that any artifact can be excluded, even a gradient of 8 mm Hg between pulmonary artery and right ventricle on a pull back tracing should be accepted as evidence of mild pulmonic stenosis

In Fig 78 an early deflection occurs during the first heart sound It is likely that *flopping of the catheter and its impact on the pulmonary arterial wall during ventricular systole* are distorting the pattern of the tracing of the pulmonary artery

In Fig 79 B, the early deflection occurring immediately after the first component of the first heart sound, may be explained by transient

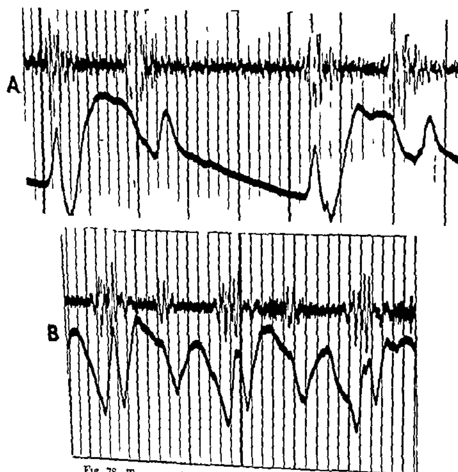


Fig 78—Tracings of pulmonary artery showing artifacts
(A) In a 29 year old woman
(B) In a 5 year-old child

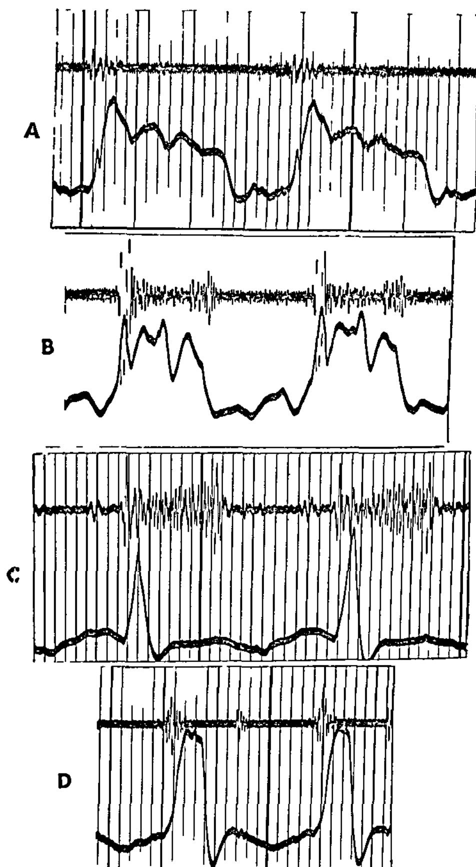
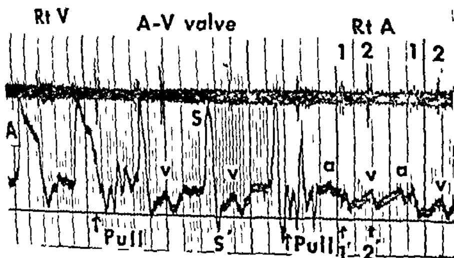


Fig 79—Four tracings of ventricular pressure which are marred by artifacts

obstruction of the lumen of the catheter by the contracting ventricular wall plus valvular vibrations The late deflection is probably due to transient obstruction of the lumen of the catheter by the contracting ventricular wall

The pressure tracing of Fig 79 A was taken while the catheter tip was located in the outflow tract of the right ventricle near the pulmonic valve The early deflection is probably caused by motion of the catheter and valvular vibrations The next deflection is probably due to the impact of the catheter tip against the pulmonary arterial wall or the wall of the right ventricular outflow tract The persisting rise in ventricular pres



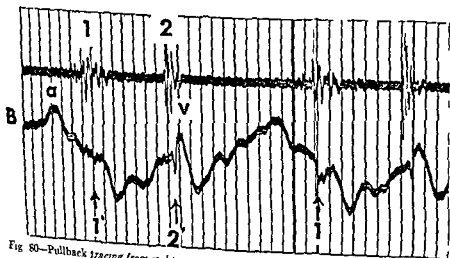


Fig 80—Pullback tracing from right ventricle to right atrium
(A) Low speed large waves are present when the tip of the catheter is near the tricuspid valve
(B) High speed catheter in the right atrium some artifacts are still present

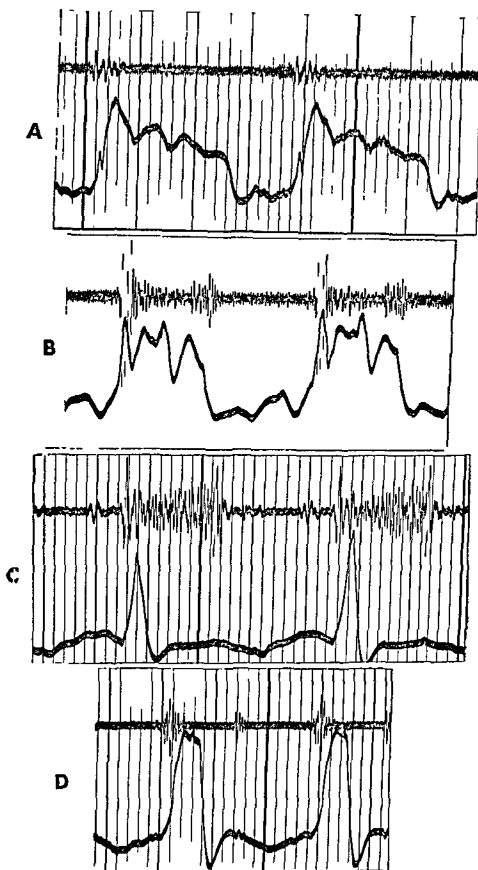


Fig 79—Four tracings of ventricular pressure which are marred by artifacts

During a pull back from the right ventricle into the right atrium, a certain number of deflections are usually recorded, as shown in Fig 79 A The deflection ss' is generally accepted as an artifact which is probably due to the impact of the tricuspid leaflets against the catheter tip rather than to the movement of the catheter in itself Subsequently the catheter tip is in the right atrium The small deflections, 1' and 2', are probably valvular in origin and are simultaneous with the heart sounds (Fig 80 B)

In Fig 81 A the superior vena caval pressure tracing has an M shaped deflection which is simultaneous with the first heart sound This deflection disappeared in a subsequent tracing recorded after slight rotation and slight pull back of the catheter This M shaped deflection may be due to partial occlusion of the catheter as a result of cardiac movements

It was previously stated that the typical pulmonary arterial "wedge" pressure tracing is seldom recorded (page 53) Theoretically when the catheter tip is wedged in a small branch of the pulmonary artery it does not move back and forth simultaneously with the cardiac movements Thus the pressure tracing recorded should be the same as or similar to those recorded in a pulmonary vein However it should be emphasized that the catheter passes through the chambers of the right heart and the

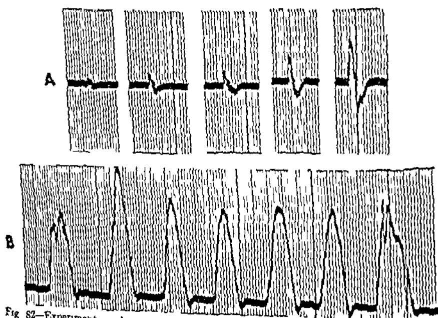


Fig 82—Experiments with a catheter showing artifacts

(A) Open catheter in water longitudinal movements records at various degrees of amplification There are diphasic complexes of proportionally increasing height

(B) Catheter closed by a small rubber balloon Application of pressure on the balloon At the time of the first and last complex the catheter loop was also moved The complexes corresponding to the motion are smaller and ... irregular in conf...

sure during the first half of diastole is probably due to the location of the tip, which is in the pulmonary artery during the first half of ventricular diastole but slips back into the right ventricular outflow tract during the remaining half

In Fig 79 C one can see an early systolic rise ending with a sharp peak, followed by an abrupt fall of pressure approaching zero after the first third of ventricular systole It is likely that the orifice of the catheter was obstructed by the contracting ventricular wall during most of ventricular systole

In Fig 79 D, *after the initial systolic rise, there is a short systolic* plateau which begins to fall just before the middle of ventricular systole and approaches zero just after the middle of systole It is possible that this was caused by occlusion of the catheter tip during the last half of ventricular systole

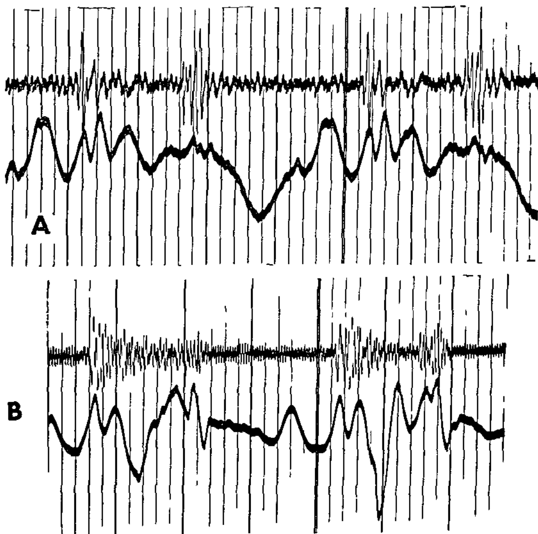


Fig 81—(A) Tracing of the superior vena cava showing artifacts at the time of the first heart sound
(B) Pulmonary arterial wedged tracing revealing multiple artifacts

CHAPTER THIRTEEN

Formulas Used in Cardiac Catheterization

The calculation of resistance in the pulmonary circulation and of the size of valvular openings presents great interest

By the application of Poiseuille equation

$$\text{resistance} = \frac{\text{pressure gradient}}{\text{rate of flow}}$$

several formulas concerning cardiovascular resistances have been derived for the evaluation of some of the unknown dynamic facts of intracardiac and pulmonary circulations The Poiseuille equation is based on a rigid tube system with continuous flow of homogeneous fluid However, the cardiovascular system has a pulsatile flow and elastic arterial walls Therefore only approximate data can be obtained from these formulas

RESISTANCE TO BLOOD FLOW

Pulmonary Arteriolar, Total Pulmonary, and Mitral Resistances

It has been suggested that the *pulmonary arteriolar resistance, or pulmonary vascular resistance* proximal to the capillary bed can be evaluated by formula (1) *

$$\text{PAR} = \frac{(\text{PA}_m - \text{PC}_m) \times 1.332}{\text{CO}} \times 0.06 = \text{dynes seconds cm}^{-5} \quad (1)$$

In this formula PAR is the pulmonary arteriolar resistance PA_m is the mean pressure of the pulmonary artery PC_m is the mean pulmonary capillary pressure' (or better mean pulmonary arterial wedge pressure) and CO is cardiac output in cc per second 1 332 is the factor for converting mm Hg to dynes per cm ²

The *total pulmonary resistance* TPR can be evaluated by formula (2)

$$\text{TPR} = \frac{\text{PA}_m \times 1.332}{\text{CO}} = \text{dynes seconds cm}^{-5} \quad (2)$$

Here the symbols are the same as in the previous formula and TPR is the total pulmonary resistance

It has been further suggested that the *resistance of the mitral valve* (MVR) can be measured through the difference between formulas (1)

* Cardiac index can be substituted for cardiac output and the unit will be dynes seconds cm ⁻⁵ per square meter of body surface

pulmonary artery, and that the size of the chambers changes from moment to moment as a result of cardiac dynamics The larger the loop made *by the catheter within the heart, the greater will be its passive movements* and the resulting artifacts (Fig 80 B) These should not be considered as actual part of the "wedge" tracing

It is generally agreed that artifacts are commonly found in the pulmonary arterial "wedge" tracing and in many of the pulmonary arterial tracings (Fig 24 and 78) The relationship between artifacts and the dynamic response of the catheter manometer systems in respect to the movements of the catheter was studied by Wood and co workers

In order to evaluate some of the artifacts which can be produced in a catheter manometer system, certain *in vitro* experiments were made in our laboratory The results are presented in Fig 82

Here, LA_{dm} is the mean diastolic pressure of the left atrium and LV_{dm} is the mean diastolic pressure of the ventricle

We have found that the incidence of *severe or pure* mitral stenosis is much rarer than that of mitral stenosis with minimal to moderate mitral insufficiency In the latter, the blood flow through the mitral valve during diastole would be greater than the calculated effective aortic forward flow which assumes no mitral insufficiency Therefore, the calculated MVR by use of formula (4) is greater than it should be, and actual mitral valve resistance is less

Further deductions from data of catheterization were drawn in order to evaluate the '*elasticity resistance' of the pulmonary vessels* This would be deduced from the data of pulse pressure and flow (cardiac output) The term 'elasticity resistance,' apparently meaning the reciprocal of distensibility is then compared to *resistance to flow* for both the pulmonary and the systemic circuits The ratio $\frac{\text{elasticity resistance}}{\text{flow resistance}}$ was found to be 3 5 for the pulmonary circulation and 1 2 for the systemic circulation These data have been criticized and cannot be accepted without further proof

Resistance of Other Stenotic Valves

Aortic stenosis The total resistance of the stenotic aortic opening can be calculated through formula (5)

$$AVR = \frac{(LV_m - BA_m) \times 1{,}332}{CO\ (\text{cc/sec}) \times \dfrac{\text{duration of total cardiac cycles (measured)}}{\text{duration of systolic ejection of same cardiac cycles (measured)}}} = \text{dynes seconds cm}^{-5} \quad (5)$$

AVR = total resistance to blood flow through the stenotic aortic valve
LV_m = left ventricular mean systolic pressure (mm Hg)
BA_m = aortic or brachial arterial mean systolic pressure (mm Hg)
CO = systemic blood flow (cc /sec)

The duration of systole is measured from the beginning of the upstroke of the aortic pressure tracing to the dicrotic notch, or from the last major vibration of the first sound to the second aortic sound in heart sound tracings Since the systolic murmur may fuse with one or both heart sounds the latter method may be unsuitable

Formula (5) gives more reliable data than another which had been previously suggested in it the blood flow through the aortic valve was calculated as though it occurred throughout the entire cardiac cycle Actually the blood flow through the stenotic aortic valve takes place only during the phase of systolic ejection

and (2) Subtracting the two formulas, one obtains formula (3)

$$MVR = \frac{PC_m \times 1332}{CO} = \text{dynes seconds cm}^{-5} \quad (3)$$

It is apparent that evaluation of mitral resistance is based entirely on the data of PC_m (or the mean values of pulmonary arterial wedge pressure) The validity of the formula is based on the assumption that the late diastolic pressure of the left ventricle is zero, a point which is questionable, especially in patients with mitral valve disease

Gorlin et al assumed a pressure of 5 mm Hg as a normal value for the diastolic level of the left ventricle The possible existence of unknown elements i e mitral insufficiency left ventricular failure, myocardial fibroelastosis of the left ventricle or constrictive pericarditis may invalidate these calculations On the other hand if the left atrial pressure and left ventricular diastolic pressure are obtained through left heart catheterization the above possibilities can usually be ruled out

It is also apparent that the blood flow through the mitral valve was computed as though it occurred throughout the entire cardiac cycle Actually, the blood flow through this valve takes place only during diastole The correct rate of flow (cc /sec) is obtained by multiplying CO by *the duration of cardiac cycles (measured) divided by the duration* of diastole of same cardiac cycles (measured) Then

$$VF = CO\ (\text{cc/sec}) \times \frac{\text{duration of cardiac cycles (measured)}}{\text{duration of diastole of same cardiac cycles (measured)}}$$

The duration of diastole can be obtained from measurements of either heart sounds or left atrial pressure tracings In heart sound tracings the duration of diastolic flow is measured from the opening snap of the mitral valve to the beginning of the first heart sound When an opening snap is not present, the duration of the interval between the beginning of the second sound and the *beginning of the first sound* (minus 0 06 to 0 08 sec) may be used In left atrial pressure tracings the duration of diastolic flow is measured from the V wave (opening of the mitral valve) to the following A V wave (closure of the mitral valve) It is also necessary to measure several cardiac cycles during one or two respiratory cycles, especially in patients with atrial fibrillation

The mitral valvular resistance can be measured through the use of formula (4) and the obtained figure is *usually confirmed by subsequent* evidence

$$MVR = \frac{(LA_{dm} - LV_d) \times 1{,}332}{CO\ (\text{cc/sec}) \times \frac{\text{duration of total cardiac cycles (measured)}}{\text{duration of diastole of same cardiac cycles (measured)}}} = \text{dynes second cm}^{-5} \quad (4)$$

(a) $F = C\,AV$
F = changes in flow rate through the orifice
C = the coefficient of orifice contraction
A = a fixed orifice
V = changes in velocity

(b) $V^2 = C_v^2\,2gh$ or $V = C_v\sqrt{2gh}$
V = changes in velocity
h = pressure mm Hg
g = gravity acceleration 980 cm /sec /sec
C = coefficient of velocity (only a certain fraction of pressure is converted to velocity)

The combination of (a) and (b) gives the area A, as in (c)

(c) $$A = \frac{F}{C_c \times C\sqrt{2gh}} = \frac{F}{C \times 44.5\sqrt{P_1 - P_2}}\ \text{cm}^2$$

F = flow rate through the orifice
C = discharge coefficient (empirical constant)
$\sqrt{2g} = \sqrt{1960} = 44.5$
$P_1 - P_2 = h$ = pressure gradient across the orifice

Mitral Valve Area

An attempt to measure mitral valve area (MVA) was made by Gorlin and Dexter through a modification of hydraulic equation (c) which gives formula (11)

$$MVA = \frac{MV_F}{31\sqrt{PC - 5}} = \frac{MV_F}{31\sqrt{LA_{dm} - LV_{dm}}} \quad (11)$$

C = discharge coefficient (empirical constant) is 0.7 in mitral stenosis
$31 = 0.7 \times 44.5$

MV_F indicates mitral valvular rate of flow in cc per second, and is obtained by dividing the *cardiac output in cc per minute* by the duration of the sum of the diastolic periods (in seconds per minute) The latter is obtained by measuring the diastolic filling per beat and multiplying by the heart rate per minute

In atrial fibrillation or sinus rhythm mitral valvular flow (MV_F) can be obtained by the following formula

$$MV_F = CO\ (\text{cc /sec}) \times \frac{\text{duration of cardiac cycles (measured)}}{\text{duration of diastoles of same cardiac cycles (measured)}} = \text{cc /sec}$$

Simultaneous pressure tracings of the left ventricle and atrium showed that filling of the left ventricle through a stenotic mitral valve may last through the early part of the ventricular tension period Therefore, duration of diastolic filling can be measured with accuracy only by either phonocardiograms or left heart catheterization

Tricuspid stenosis By substituting right atrial mean diastolic pressure (RA_{dm}) for the corresponding left atrial data (LA_{dm}) in formula (4) and right ventricular mean diastolic pressure (RV_{dm}) for the corresponding left ventricular data (LV_{dm}) in formula (4), the resistance of the tricuspid valve can be deduced, as in formula (6)

$$TVR = \frac{(RA_{dm} - RV_{dm}) \times 1332}{CO \times \dfrac{\text{duration of total cardiac cycles (measured)}}{\text{duration of diastoles of same cycles (measured)}}} = \text{dynes seconds cm}^{-5} \quad (6)$$

Pulmonic stenosis The total resistance of the pulmonic opening ir respective of whether there is valvular, infundibular, or a complex type of stenosis, can be calculated through formula (7)

$$PVR = \frac{(RV_{m} - PA_{m}) \times 1{,}332}{CO \times \dfrac{\text{duration of total cardiac cycles (measured)}}{\text{duration of systolic ejections of same cycles (measured)}}} = \text{dynes seconds cm}^{-5} \quad (7)$$

PVR = total resistance to blood flow through the stenotic pulmonic valve
RV_{m} = right ventricular mean systolic pressure mm Hg
PA_{sm} = pulmonary arterial mean systolic pressure mm Hg
CO = pulmonary blood flow (cc /sec)

The resistance of the infundibulum (PIR) in a complex type of stenosis can be calculated by using formula (8)

$$PIR = \frac{(RV_{m} - RVI_{m}) \times 1332}{CO \times \dfrac{\text{duration of total cardiac cycles (measured)}}{\text{duration of systolic ejections of same cycles (measured)}}} = \text{dynes seconds cm}^{-5} \quad (8)$$

RVI_{m} = infundibular mean systolic pressure in mm Hg

Likewise, the resistance of the stenotic pulmonary valve in a complex type of stenosis can be obtained by formula (9)

$$RPV = \frac{(RVI_{m} - PA_{m}) \times 1332}{CO \times \dfrac{\text{duration of total cardiac cycles (measured)}}{\text{duration of systolic ejections of same cycles (measured)}}} = \text{dynes seconds cm}^{-5} \quad (9)$$

The calculated *peripheral resistance* can be obtained by using formula (10)

$$\text{Peripheral resistance} = \frac{BA_{m} \times 1{,}332}{CO} = \text{dynes second cm}^{-5} \quad (10)$$

Formulas for calculating valvular area are derived by the application of the two well known hydraulic equations (a) and (b)

(a) $F = C A V$

F = changes in flow rate through the orifice
C = the coefficient of orifice contraction
A = a fixed orifice
V = changes in velocity

(b) $V^2 = C_v 2^2 gh$ or $V = C_v\sqrt{2gh}$

V = changes in velocity
h = pressure mm Hg
g = gravity acceleration 980 cm /sec /sec
C = coefficient of velocity (only a certain fraction of pressure is converted to velocity)

The combination of (a) and (b) gives the area A, as in (c)

$$\text{(c)}\ A = \frac{F}{C \times C\sqrt{2gh}} = \frac{F}{C \times 44.5\sqrt{P_1 - P_2}}\ \text{cm}^2$$

F = flow rate through the orifice
C = discharge coefficient (empirical constant)
$\sqrt{2g} = \sqrt{1960} = 44.5$
$P_1 - P_2 = h$ = pressure gradient across the orifice

Mitral Valve Area

An attempt to measure mitral valve area (MVA) was made by Gorlin and Dexter through a modification of hydraulic equation (c) which gives formula (11)

$$MVA = \frac{MV_F}{31\sqrt{PC - 5}} = \frac{MV_F}{31\sqrt{LA_{dm} - LV_{dm}}} \qquad (11)$$

C = discharge coefficient (empirical constant) is 0.7 in mitral stenosis
$31 = 0.7 \times 44.5$

MV_F indicates mitral valvular rate of flow in cc per second, and is obtained by dividing the *cardiac output in cc per minute* by the duration of the sum of the diastolic periods (in seconds per minute) The latter is obtained by measuring the diastolic filling per beat and multiplying by the heart rate per minute

In atrial fibrillation or sinus rhythm mitral valvular flow (MV_F) can be obtained by the following formula

$$MV_F = CO\ (\text{cc /sec}) \times \frac{\text{duration of cardiac cycles (measured)}}{\text{duration of diastoles of same cardiac cycles (measured)}} = \text{cc /sec}$$

Simultaneous pressure tracings of the left ventricle and atrium showed that filling of the left ventricle through a stenotic mitral valve may last through the early part of the ventricular tension period Therefore, duration of diastolic filling can be measured with accuracy only by either phonocardiograms or left heart catheterization

The number 31 used in formula (11) is an *empirical constant which is supposed to correct for anomalies of discharge through the orifice and errors in calculating the diastolic filling period, and convert mm Hg to cm H_2O The figure 5 used in formula (11) is acceptable as the estimated level of left ventricular mean diastolic pressure

Aortic Valve Area

The cross sectional area of the aortic valve (AVA) is calculated by Gorlin's formula (12)

$$AVA = \frac{AVF}{C \times 44\ 5\sqrt{LV_m - BA_m}} = cm^2 \qquad (12)$$

AVF = *Aortic valve flow in cc per second*

$$\left(\frac{\text{cardiac output}}{\text{systolic ejection period in seconds per minute}}\right)$$

LV_m = *Left ventricular systolic ejection mean pressure in mm Hg*
BA_m = Brachial arterial systolic mean pressure in mm Hg
C = empirical constant to be derived

Systolic ejection period in seconds per minute can be obtained by

$$\frac{\text{total duration in seconds of systolic ejection periods of cardiac cycles (measured)}}{\text{total duration in seconds of same cardiac cycle (measured)}} \times 60$$

A single systolic ejection period can be measured on the aortic and left ventricular pressure tracings from the point where the beginning of the aortic upstroke (or from the point at which left ventricular pressure *equals arterial diastolic pressure) to the point of the aortic incisura or that* where left ventricular pressure falls below the arterial dicrotic notch

The value of the empirical constant C has not yet been derived because left heart catheterization has been in use for only a few years Patients studied by us have not been subjected to either direct vision valvotomy or autopsy *for measuring the aortic valvular cross sectional area* At present, we use an arbitrary value of 1 0 for C The figure may be too high because the conversion factor of 1 17 is included in C and the actual discharge coefficient ($C_v \times C_c$) of the orifice may be lower than 0 85

Pulmonic Valve Area

The cross sectional area of the pulmonic valve (PVA) is calculated by Gorlin's formula (13)

$$PVA = \frac{PVF}{C \times 44\ 5\ RV_m - PA_m} = cm^2 \qquad (13)$$

PVF = pulmonic valve flow in cc per second

$$\left(\frac{\text{cardiac output}}{\text{systolic ejection period in seconds per minute}}\right)$$

RV_m = right ventricular mean systolic ejection pressure in mm Hg
PA_{sm} = pulmonary arterial mean systolic pressure in mm Hg

Systolic ejection period in seconds per minute can be obtained by

$$\frac{\text{total duration in seconds of systolic ejection period of cardiac cycles (measured)}}{\text{total duration in seconds of same cardiac cycles (measured)}} \times 60$$

A single systolic ejection period can be measured on the right ventricular pressure tracing from the point where the right ventricular pressure equals pulmonary pressure to the point where right ventricular pressure equals the pulmonary arterial dicrotic notch

The measurement of pulmonary arterial mean systolic pressure should be carefully done because multiple artifacts are usually present in the pulmonary arterial pressure tracing One should not consider the early diastolic rebound as the systolic pressure (Chapter eleven)

Tricuspid Valve Area

The cross sectional area of the tricuspid valve (TVA) is calculated by formula (14), a modification of Gorlin s formula

$$TVA = \frac{TVF}{C \times 44.5 \sqrt{RA_{dm} - RV_{dm}}} = cm^2 \qquad (14)$$

TVF = tricuspid valve flow in cc per sec

$$\left(\frac{\text{cardiac output}}{\text{diastolic filling of the right ventricle per minute}}\right)$$

RA_{dm} = right atrial diastolic mean pressure in mm Hg
RV_{dm} = right ventricular diastolic mean pressure in mm Hg

The value of the empirical constant C has not been determined as yet Since no correction for diastolic filling is necessary, C is greater than 0 7 Assuming the discharge coefficient ($C_c \times C_v$) to be 0 85, the value of 1 0 was used by the Gorlins This figure C may be changed with future collection of autopsy data

Those interested in the formulas for calculating the diameter of a patent ductus arteriosus or the areas of interatrial or interventricular septal defects should consult a publication by the Gorlins

Work Performed by the Ventricles

The work performed by the right ventricle (RVWD) can be calculated by formulas (15) or (15a) which are simple and practical Formula (16) is based on the metric system and is more accurate

$$RVWD = CO\,(PA_{m} - RA_{m}) \times 1{,}332 = \text{dynes cm/sec} \qquad (15)$$

CO = cardiac output cc /sec
PA_m = pulmonary arterial mean systolic pressure mm Hg
RA_m = right atrial mean pressure (may be omitted in order to obtain an approximate value)
1,332 = factor to convert mm Hg to dynes per cm²

Cardiac output = pulmonary output and cardiac index = pulmonary index in the absence of shunts

The number 31 used in formula (11) is an empirical constant which is supposed to correct for anomalies of discharge through the orifice and errors in calculating the diastolic filling period, and convert *mm Hg to* cm H_2O The figure 5 used in formula (11) is acceptable as the estimated level of left ventricular mean diastolic pressure

Aortic Valve Area

The cross sectional area *of* the *aortic valve* (AVA) *is calculated by* Gorlin's formula (12)

$$AVA = \frac{AVF}{C \times 44\,5\sqrt{LV_m - BA_m}} = cm^2 \qquad (12)$$

AVF = Aortic valve flow in cc per second $\left(\frac{\text{cardiac output}}{\text{systolic ejection period in seconds per minute}}\right)$

LV_m = Left ventricular systolic ejection mean pressure in mm Hg

BA_m = Brachial arterial systolic mean pressure in mm Hg

C = empirical constant to be derived

Systolic ejection period in seconds per minute can be obtained by

$$\frac{\text{total duration in seconds of systolic ejection periods of cardiac cycles (measured)}}{\text{total duration in seconds of same cardiac cycle (measured)}} \times 60$$

A single systolic ejection period can be measured on the aortic and left ventricular pressure tracings from the point where the beginning of the aortic upstroke (or from the point at which left ventricular pressure equals arterial diastolic pressure) to the point of the aortic incisura or that where left ventricular pressure falls below the arterial dicrotic notch

The value of the empirical constant C has not yet been derived because left heart catheterization has been in use for only a few years *Patients* studied by us have not been subjected to either direct vision valvotomy or autopsy for measuring the aortic valvular cross sectional area At present, we use an arbitrary value of 1 0 for C The figure may be too *high because the conversion factor of* 1 17 *is included in* C and the actual discharge coefficient ($C_v \times C_c$) of the orifice may be lower than 0 85

Pulmonic Valve Area

The cross sectional area of the pulmonic valve (PVA) is calculated by Gorlin's formula (13)

$$PVA = \frac{PVF}{C \times 44\,5\,RV_m - PA_m} = cm^2 \qquad (13)$$

PVF = pulmonic valve flow in cc per second $\left(\frac{\text{cardiac output}}{\textit{systolic ejection period in seconds per minute}}\right)$

RV_m = right ventricular mean systolic ejection pressure in mm Hg

PA_m = pulmonary arterial mean systolic pressure in mm Hg

sample is collected, then the oxygen content of the sample will be lower than it should (hepatic blood instead of mixed lower caval blood) If the tip is below the orifice of the hepatic veins then the oxygen content of the sample is higher

(b) The inferior vena cava carries about 55 to 60 per cent of the total amount of blood into the right atrium while the superior vena cava carries 40 to 45

(c) The coronary sinus carries about 5 per cent of the total output, consisting of blood with a very low oxygen content (3 to 4 volumes per cent) This blood will be mixed in the lower part of the right atrium and, more thoroughly, in the right ventricle

(d) Mixed venous blood is more thoroughly mixed in the pulmonary artery than in the right atrium or ventricle of patients having no evidence of shunt

(e) The following data are considered by us to be evidence of left to right shunt between the atria

(1) The average oxygen content of the right atrium should be at least 1.5 volumes per cent higher than the average of the blood samples of the two venae cavae

(2) The oxygen content of a single right atrial blood sample should be at least 3 volumes per cent higher than the average of the blood samples of the two venae cavae

(3) The oxygen content of two or more right atrial blood samples should be at least 4 volumes per cent higher than that of either caval sample

(f) In the absence of primary alveolar changes interfering with gas diffusion a 95 per cent oxygen saturation in the blood of the pulmonary veins or left atrium is considered normal Several factors prevent the theoretical full saturation (100 per cent)

(g) In spite of conflicting statements we accept pulmonary venous return as identical with pulmonary arterial flow

In *isolated atrial septal defect* with left to right shunt the data obtained through catheterization are more reliable than in isolated ventricular septal defect or patent ductus arteriosus because the blood obtained from the pulmonary artery is already thoroughly mixed

If there is evidence of double left to right shunt through both an atrial and a ventricular septal defect the calculated figures of flow for the single shunts are not too reliable However the total amount of left to right shunt is as reliable as in an isolated ventricular septal defect

In the presence of a large left to right shunt through a patent ductus arteriosus the presence of a high ventricular septal defect can be ruled out only with difficulty because one cannot exclude penetration of shunted blood with high oxygen content from the pulmonary artery into the right ventricle because of pulmonic insufficiency

Calculation of the severity of a pure right to left shunt or a bidirectional shunt in atrial septal defects is easily made

Since

$$1 \text{ erg} = 1 \text{ dyne} \times \text{cm}$$
$$1 \text{ joule} = 10^7 \text{ dynes} \times \text{cm},$$

by substituting joules and omitting RA_m, the formula may be simplified as follows

$$RVWD = \frac{CO \times PA_m \times 1.332}{10^7} = \text{joules/sec} \quad (15a)$$

A more accurate formulation would be

$$RVWD = \frac{(CI \times 1.055)[PA_m - RA_m] \times 13.6}{1\,000} = \text{KgM/min/M}^2 \quad (16)$$

KgM (meter)/min/m² (square meters of body surface)
CI* = cardiac index in liters/min/M²
1.055 = specific gravity of the blood
13.6 = specific weight of mercury

It can be further simplified by omitting RA_m

$$RVWD = \frac{(CI \times 1.055)\ PA_m \times 13.6}{1\,000} = \text{KgM/min/M}^2 \quad (16a)$$

CI = cardiac index of pulmonary flow in liters/min/m²

By substituting brachial or femoral mean systolic pressure (BA_{sm}) for PA_m, and LA_m for RA_m, the work done by the left ventricle (LV) can be also evaluated

The calculation of the amount of blood flowing through shunts requires knowledge of several data including oxygen consumption by the patient and the oxygen contents and pressures of the various cardiovascular chambers and of the peripheral blood For this reason even though this study was particularly concerned with the pressures and pulses of the various chambers, the following pages will refer to oxygen determination data Thus most of the formulas needed in cardiac catheterization will be included in a single, brief chapter

Intracardiac Shunts

The evaluation of intracardiac shunts was attempted soon after right heart catheterization was introduced as a diagnostic procedure Much of the following outline is universally accepted

(a) The oxygen contents of the superior and inferior venae cavae usually differ by one to three volumes per cent The blood sample of the inferior vena cava should be taken when the tip of the catheter is just below the diaphragm If this tip advances to the orifice of the hepatic veins (which usually carry blood with a lower oxygen content) while the

$$PBF = \frac{O_2 \times 100}{BA - PA} = \text{cc/min} \quad (3)$$

The left to right shunt can be easily obtained by subtracting systemic blood flow from pulmonary blood flow as in formula (4)

$$Y_A = PBF - SBF = \text{cc/min} \quad (4)$$

Also, the left-to-right shunt can be estimated from the total O_2 content of blood passing through the right atrium in one minute as in formula (5)

$$SBF \times SIVC + Y_A \times BA = SBF \times RA_{ave} + Y_A \times RA_{ave} \quad (5)$$

This equation can be simplified as follows

$$Y_A(BA - RA_{ave}) = SBF(RA_{ave} - SIVC) \text{ Next} \quad (5a)$$

$$Y_A = \frac{SBF(RA_{ave} - SIVC)}{(BA - RA_{ave})} = \text{cc/min} \quad (5b)$$

We have studied a patient with atrial septal defect and right to-left shunt by simultaneous catheterization of both the right and left heart by inserting two needles one into the left atrium and another into the right *Simultaneous* pressure recordings of both atria and ventricles were made by means of polyethylene catheters Blood samples taken in the left atrium showed a significant decrease in oxygen content but there was no *further decrease* in the left ventricle and the brachial artery This procedure certainly located the site of right to-left shunt precisely However, it is only of academic interest

In the right ventricle Left to right shunt between ventricles is presumed if SIVC = RA_{ave} and RV_{ave} = PA, but $RV_{ave} > RA_{ave}$ (by more than 1 0 vol per cent)

The left to right shunt can be estimated by using either formulas (6) or (7)

$$Y = PBF - SBF = \text{cc/min} \quad (6)$$

$$Y_V = \frac{SBF(LA - RA_{ave})}{(BA - PA)} = \frac{SBF(RV_{ave} - RA_{ave})}{(BA - RV_{ave})} = \text{cc/min} \quad (6a)\ (6b)$$

Between aorta and pulmonary artery Left to right shunt between the larger arteries is presumed if SIVC = RA_{ave} = RV_{ave}, but $PA_{ave} > RV$ (by more than 1 0 vol per cent)

A left to right shunt through a patent ductus arteriosus or any other aorto pulmonary communication can be estimated by formulas (7) or (7a)

$$Y_{PA} = PBF - SBF = \text{cc/min} \quad (7)$$

$$Y_{PA} = SBF\frac{(PA_{ave} - RA_{ave})}{(BA - PA_{ave})} = \text{cc/min} \quad (7a)$$

FORMULAS FOR CALCULATION OF A SHUNT*

Absence of Shunts

In the *absence of shunts*, according to Fick, the cardiac output (or estimated systemic blood flow SBF, which equals pulmonary arterial blood flow PFB) is calculated by dividing the oxygen consumption by the difference between arterial and mixed venous bloods (pulmonary arterial sample)

$$SBF = PBF = \frac{O_2 \times 100}{BA - PA} = cc/min \qquad (1)$$

Left to Right Shunts

In the right atrium *Anomalous pulmonary venous return into the right atrium, or atrial septal defect with left to right shunt,* is suspected when RA_{avg} > SIVC (by 1 5 vol per cent or more) and RA_{avg} = PA

Anomalous pulmonary venous return into either vena cava is suspected when the difference in O_2 content is more than 4 5 vol per cent between inferior and superior venae cavae If the oxygen content of the superior vena cava is 2 or more volumes per cent higher than that of the higher location in the superior cava or of the innominate veins, partial anomalous pulmonary venous return into the superior cava is suspected

The systemic blood flow in the first instance is calculated through formula (2), and the pulmonary flow through formula (3)

$$SBF = \frac{O_2 \times 100}{BA - SIVC} = cc/min \qquad (2)$$

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* The formulas for shunts use the following symbols and abbreviations
O_2 = O_2 consumption cc/min
SBF = systemic blood flow, cc/min
PBF = pulmonary blood flow cc/min
EPBF = effective pulmonary blood flow cc/min
$PI_{PA\,RV}$ = pulmonary insufficiency cc/min
SIVC = O_2 content of both vena cava vol per cent (SIC × 0 40 + IVC × 0 60)
RA_{avg} = O_2 content of average right atrium vol per cent
RV_{avg} = O_2 content of average right ventricle vol per cent
RV_{M+H} = O_2 content of average of mid and high right ventricle vol per cent
PA = O_2 content of pulmonary artery vol per cent
PA_{avg} = O_2 content of average left and right pulmonary arteries vol per cent (in patent ductus arteriosus)
LA = O_2 content of left atrium vol per cent
BA = O_2 content of brachial artery vol per cent
PV = O_2 content of pulmonary vein vol per cent (assumed to be 95 per cent saturated)
P_{RA} = right atrial pressure mm Hg
P_{RV} = right ventricular pressure mm Hg
P_{PA} = pulmonary arterial pressure mm Hg
P_{BA} = brachial arterial pressure mm Hg
Y = left to right shunt (YA = atrial level YV = ventricular level)
Z = right to left shunt (ZA = atrial level ZV = ventricular level)

In cases of large left to right shunt due to patent ductus arteriosus (without any ventricular septal defect), one may find that the oxygen content of the right ventricular sample (especially if obtained from the outflow tract) is somewhat higher (1 0 vol per cent) than that of the samples of the right atrium and of the inflow tract (or lower part) of the right ventricle In such cases we use RA_{s} instead of RV_{s}

At times it is difficult to decide whether or not there is an additional high ventricular defect or pulmonic insufficiency

Pulmonary insufficiency If *pulmonary insufficiency* is suspected in *a case with patent ductus arteriosus* the amount of regurgitation can also be calculated but the result obtained from the average of high and mid right ventricular samples gives only a rough estimate formula (12)

$$PI_{PV,RV} = \frac{SBF(RV_{H+M} - RA_{s}) \times 100}{(PA_{s} - RV_{H+M})} = \text{cc/min} \qquad (12)$$

Right to Left Shunts

Atrial septal defect with or without pulmonic stenosis This shunt can be calculated by comparing the pressures of the right ventricle and brachial artery simultaneously recorded The O_2 saturation of BA is below 92 per cent

If $P_{RVS} < P_{BAS}$ there is no right to left shunt provided that overriding of the aorta is ruled out by angiocardiography

If $P_{RVS} > P_{PAS}$ pulmonic stenosis is present but still there is no shunt

If $P_{RAM} = P_{RVD}$ and is slightly elevated (6 to 10 mm Hg) and moreover BA sat < 92 per cent and $RA_{s} = RV_{s} = LA$ then there is evidence of a right to left shunt through an atrial septal defect Such a shunt can be calculated from formula (13b) derived from formulas (13) and (13a)

$$SBF = \frac{O_2 \times 100}{BA - PA} = \text{cc/min} \qquad (13)$$

$$PBF = \frac{O_2 \times 100}{LA - LA} = \text{cc/min} \qquad (13a)$$

$$Z = SBF - PBF = \text{cc/min} \qquad (13b)$$

In formulas (14) and (14b) the calculation of right to left shunt is based on the total O_2 content of the blood which contributes to the mixing in the left atrium in one minute It is assumed that the pulmonary venous blood is 95 per cent saturated with oxygen

$$PBF \times BA + Z \times BA = PBF \times LA + Z \times PA \qquad (14)$$

$$Z(BA - PA) = PBF(LA - BA) \qquad (14a)$$

$$Z = \frac{PBF(LA - BA)}{(BA - PA)} = \text{cc/min} \qquad (14b)$$

Ventricular septal defect with or without pulmonic stenosis (tetralogy of Fallot or Eisenmenger complex)

The pulmonary blood flow (PBF) of formula (7) should be calculated by formula (8) from the average of samples collected from both the left and the right pulmonary arteries. It is preferable to obtain 2 samples of blood from each stem of the pulmonary artery (4 in all).

$$PBF = \frac{O_2 \times 100}{(BA - PA_{avg})} = cc/min \qquad (8)$$

If only one pulmonary random blood sample is obtained near the orifice of the patent ductus, it may have a much higher oxygen content, and the pulmonary blood flow thus calculated may become as high as 40 liters/min. This is obviously incorrect.

In formula (7a), the use of RA_{avg} instead of RV_{avg} is based on the fact that there may be pulmonary regurgitation which would increase the oxygen content of the right ventricular samples, especially in the upper part of the ventricle.

With both atrial and ventricular defect.

$RA_{avg} > SIVC$ (by more than 1.5 vol. per cent) while
$RV_{avg} > RA_{avg}$ (by more than 1.0 vol. per cent) and
$RV_{avg} = PA$

Y_A, the left to right shunt through the atrial defect, can be calculated by formula (5b); Y_V, the left to right shunt through the ventricular defect, can be calculated by formula (9) or (9a).

$$Y_V = PBF - (SBF + Y_A) = cc/min \qquad (9)$$

$$Y_V = \frac{(SBF + Y_A)(PA - RA_{avg})}{(BA - PA)} = cc/min \qquad (9a)$$

Y_{A+V}, the total left to right shunt, can be obtained by formulas (10) and (10a).

$$Y_{A+V} = PBF - SBF = cc/min \qquad (10)$$

$$Y_{A+V} = \frac{SBF(PA - SIVC)}{(BA - PA)} = cc/min \qquad (10a)$$

With ventricular septal defect and patent ductus arteriosus. When both are present, the following will be true:

$SIVC = RA_{avg}$
$RV_{avg} > RA_{avg}$ (by more than 1.0 vol. per cent) and
$PA_{avg} > RV_{avg}$ (by more than 1.5 vol. per cent)

Y_V, the left to right shunt through the ventricular defect, can be calculated by formula (6b).

Y_{PA}, the left to right shunt through a patent ductus can be calculated by formulas (11) and (11a). Formula (11) is preferred for its simplicity.

$$Y_{PA} = PBF - (SBF + Y_V) = cc/min \qquad (11)$$

$$Y_{PA} = \frac{(SBF + Y_V)(PA_{avg} - RA_{avg})}{BA - PA_{avg}} = cc/min \qquad (11a)$$

In cases of large left to right shunt due to patent ductus arteriosus (without any ventricular septal defect), one may find that the oxygen content of the right ventricular sample (especially if obtained from the outflow tract) is somewhat higher (1 0 vol per cent) than that of the samples of the right atrium and of the inflow tract (or lower part) of the right ventricle In such cases we use RA , instead of RV_{a} ,

At times it is difficult to decide whether or not there is an additional high ventricular defect or pulmonic insufficiency

Pulmonary insufficiency If *pulmonary insufficiency* is suspected in a case *with patent ductus arteriosus*, the amount of regurgitation can also be calculated but the result obtained from the average of high and mid right ventricular samples gives only a rough estimate formula (12)

$$PI_{PA-RV} = \frac{SBF(RV_{H+M} - RA\ \ ) \times 100}{(LA\ \ - RV_{H+M})} = cc/min \tag{12}$$

Right to Left Shunts

Atrial septal defect with or without pulmonic stenosis This shunt can be calculated by comparing the pressures of the right ventricle and brachial artery simultaneously recorded The O_2 saturation of BA is below 92 per cent

If $P_{RVS} < P_{BAS}$ there is no right to left shunt provided that overriding of the aorta is ruled out by angiocardiography

If $P_{RVS} > P_{BAS}$ pulmonic stenosis is present but still there is no shunt

If $P_{RAM} = P_{RVD}$ and is slightly elevated (6 to 10 mm Hg) and moreover BA sat < 92 per cent and RA $=$ RV $=$ PA then there is evidence of a right to left shunt through an atrial septal defect Such a shunt can be calculated from formula (13b) derived from formulas (13) and (13a)

$$SBF = \frac{O\ \ \times 100}{BA - PA} = cc/min \tag{13}$$

$$PBF = \frac{O_2 \times 100}{LA - PA} = cc/min \tag{13a}$$

$$Z = SBF - PBF = cc/min \tag{13b}$$

In formulas (14) and (14b) the calculation of right to left shunt is based on the total O_2 content of the blood which contributes to the mixing in the left atrium in one minute It is assumed that the pulmonary venous blood is 95 per cent saturated with oxygen

$$PBF \times BA + Z \times BA = PBF \times LA + Z \times PA \tag{14}$$

$$Z(BA - PA) = PBF(LA - BA) \tag{14a}$$

$$Z = \frac{PBF(LA - BA)}{(BA - PA)} = cc/min \tag{14b}$$

Ventricular septal defect with or without pulmonic stenosis (tetralogy of Fallot or Eisenmenger complex)

The pulmonary blood flow (PBF) of formula (7) should be calculated by formula (8) from the average of samples collected from both the left and the right pulmonary arteries. It is preferable to obtain 2 samples of blood from each stem of the pulmonary artery (4 in all).

$$PBF = \frac{O_2 \times 100}{(BA - PA_{avg})} = cc/min \qquad (8)$$

If only one pulmonary random blood sample is obtained near the orifice of the patent ductus, it may have a much higher oxygen content, and the pulmonary blood flow thus calculated may become as high as 40 liters/min. This is obviously incorrect.

In formula (7a), the use of RA_{avg} instead of RV_{avg} is based on the fact that there may be pulmonary regurgitation which would increase the oxygen content of the right ventricular samples, especially in the upper part of the ventricle.

With both atrial and ventricular defect

$RA_{avg} > SIVC$ (by more than 1.5 vol per cent) while
$RV_{avg} > RA_{avg}$ (by more than 1.0 vol per cent) and
$RV_{avg} = PA$

Y_A, the left to right shunt through the atrial defect, can be calculated by formula (5b). Y_V, the left to right shunt through the ventricular defect, can be calculated by formula (9) or (9a).

$$Y_V = PBF - (SBF + Y_A) = cc/min \qquad (9)$$

$$Y_V = \frac{(SBF + Y_A)(PA - RA_{avg})}{(BA - PA)} = cc/min \qquad (9a)$$

Y_{A+V}, the total left to right shunt, can be obtained by formulas (10) and (10a).

$$Y_{A+V} = PBF - SBF = cc/min \qquad (10)$$

$$Y_{A+V} = \frac{SBF(PA - SIVC)}{(BA - PA)} = cc/min \qquad (10a)$$

With ventricular septal defect and patent ductus arteriosus. When both are present, the following will be true:

$SIVC = RA_{avg}$
$RV_{avg} > RA_{avg}$ (by more than 1.0 vol per cent) and
$PA_{avg} > RV_{avg}$ (by more than 1.5 vol per cent)

Y_V, the left to right shunt through the ventricular defect, can be calculated by formula (6b).

Y_{PA}, the left to right shunt through a patent ductus can be calculated by formulas (11) and (11a). Formula (11) is preferred for its simplicity.

$$Y_{PA} = PBF - (SBF + Y_V) = cc/min \qquad (11)$$

$$Y_{PA} = \frac{(SBF + Y_V)(PA_{avg} - RA_{avg})}{BA - PA_{avg}} = cc/min \qquad (11a)$$

$$Z\,(RA - SIVC) = PBF(LA - RA) + Y\,(RA - LA) \qquad (18b)$$

$$Z = \frac{PBF(LA - RA) + Y(RA - LA)}{(RA - SIVC)} \qquad (18c)$$

Substitute (18c) into (17c) and formula (19) is derived

$$Y = \frac{SBF(PA - SIVC) + \left(\frac{PBF(LA - RA) + Y(RA - LA)}{RA - SIVC}\right)(SIVC - PA)}{(LA - PA)} \qquad (19)$$

Knowing pulmonary flow, effective pulmonary flow, and systemic flow, we can use Bing's formulas (20) and (21) for calculating right to left and left to right shunts The "effective pulmonary flow" EPBF will be described below

$$Y = PBF - EPBF = \text{cc/min} \qquad (20)$$

$$Z = SBF - EPBF = \text{cc/min} \qquad (21)$$

Eisenmenger complex In this case SIVC = RA_{O_2}, and RV_{O_2} > RA_{O_2}, but RV_{O_2} = PA

As in atrial septal defect with bidirectional shunt, we can use formulas (15), (16) (17c) (18c), (19) or (20) and (21) in order to calculate the left to right (Y) and right to left (Z) shunts

In order to obtain a more reliable result it is advisable to substitute the SIVC for RA_{O_2}

The calculation of the "effective pulmonary blood flow" EPBF was advocated by Bing, and it is defined as the amount of mixed venous blood which, having returned to the heart from the systemic circulation, eventually reaches the pulmonary capillaries Normally, the effective pulmonary blood flow equals pulmonary arterial flow, except when bidirectional shunts are present—as in some cases of atrial septal defect complicated by pulmonic stenosis and in cases of the Eisenmenger complex

Formula (22) is useful in the former cases and (23) in the latter

$$EPBF = \frac{O \times 100}{PV - SIVC} = \text{cc/min} \qquad (22)$$

$$EPBF = \frac{O \times 100}{LA - RA} = \text{cc/min} \qquad (23)$$

Pulmonic stenosis especially in cases of tetralogy of Fallot, may be accompanied by large collateral (bronchial) circulation or by a patent ductus arteriosus In such cases total pulmonary blood flow or pulmonary capillary blood flow is larger than the main pulmonary arterial blood flow The former can only be calculated by indirect means, and the result is only a rough estimate

If $RA_{avg} = RV_{avg} = PA$ there is no left to right shunt

If P_{RA} mean is not elevated (less than 6 mm Hg) there is probably no right to left shunt through an additional atrial septal defect

If BA sat < 92 per cent there is right to left shunt

The systemic blood flow, pulmonary blood flow, and right to left shunt can be likewise calculated by formulas (13), (13a), and (13b) or (14)

Patent ductus arteriosus with pulmonic hypertension The calculations of the right to left shunt can only be based on certain assumptions (1) The shunt takes place from the pulmonary artery to the aorta at a point (ductus) which is below the opening of the left carotid artery and above that of the left subclavian artery (2) A minor portion of the shunted blood goes back to the aortic arch and enters the innominate artery and the two carotids, while a major portion is distributed between left subclavian and descending aorta This second assumption, although used by some, is definitely unwarranted because, in some cases, no evidence of low oxygen saturation can be found in either the left arm or the left earlobe Therefore, as we have no accurate method of ascertaining this distribution of blood, accurate mathematical calculation of the amount of shunt is impossible

Bidirectional Shunts (Left to Right plus Right to Left Shunts)

Atrial septal defect with or without pulmonic stenosis The calculations are based on the data of the total O_2 content which contributes to the mixing in the left and right atria in one minute It is assumed that $RA_{avg} = RV_{avg} = PA$ and that $LA = 95$ per cent O_2 saturation Formulas (15) and (16) are used

$$PBF = \frac{O_2 \times 100}{LA - PA} \text{ cc/min} \quad (15)$$

$$SBF = \frac{O_2 \times 100}{BA - SIVC} \text{ cc/min} \quad (16)$$

In the *right atrium*, the amount of blood which contributes to the mixing in one minute is calculated by formulas (17), (17a), (17b), and (17c)

$$(SBF - Z)SIVC + Y \times LA = (SBF - Z + Y) \times PA \quad (17)$$

$$SBF \times SIVC - Z \times SIVC + Y \times LA = SBF \times PA - Z \times PA + Y \times PA \quad (17a)$$

$$Y(LA - PA) = SBF(PA - SIVC) + Z(SIVC - PA) \quad (17b)$$

$$Y = \frac{SBF(PA - SIVC) + Z(SIVC - PA)}{LA - PA} \quad (17c)$$

In the *left atrium*, the amount of blood which contributes to the mixing in one minute is calculated by formulas (18), (18a), (18b), and (18c)

$$(PBF - Y)LA + Z \times SIVC = (PBF - Y + Z) \times BA \quad (18)$$

$$PBF \times LA - Y \times LA + Z \times SIVC = PBF \times BA - Y \times BA + Z \times BA \quad (18a)$$

$$Z\,(BA - SIVC) = PBF(LA - BA) + X\,(BA - LA) \qquad (18b)$$

$$Z = \frac{PBF(LA - BA) + X(BA - LA)}{(BA - SIVC)} \qquad (18c)$$

Substitute (18c) into (17c) and formula (19) is derived

$$X = \frac{SBF(PA - SIVC) + \left(\frac{PBF(LA - BA) + X(BA - LA)}{BA - SIVC}\right)(SIVC - LA)}{(LA - PA)} \qquad (19)$$

Knowing pulmonary flow, effective pulmonary flow, and systemic flow, we can use Bing's formulas (20) and (21) for calculating right to left and left to right shunts The "effective pulmonary flow" EPBF will be described below

$$X = PBF - EPBF = cc/min. \qquad (20)$$

$$Z = SBF - EPBF = cc/min \qquad (21)$$

Eisenmenger complex In this case SIVC = $RA_{a.v.}$ and $RV_{a.v.} > RA_{a.v.}$, but $RV_{a.v.}$ = PA

As in atrial septal defect with bidirectional shunt, we can use formulas (15), (16) (17c) (18c) (19) or (20) and (21) in order to calculate the left to-right (X) and right to left (Z) shunts

In order to obtain a more reliable result it is advisable to substitute the SIVC for $RA_{a.v.}$

The calculation of the "effective pulmonary blood flow" EPBF was advocated by Bing and it is defined as the amount of mixed venous blood which having returned to the heart from the systemic circulation, eventually reaches the pulmonary capillaries Normally, the effective pulmonary blood flow equals pulmonary arterial flow except when bidirectional shunts are present—as in some cases of atrial septal defect complicated by pulmonic stenosis, and in cases of the Eisenmenger complex

Formula (22) is useful in the former cases and (23) in the latter

$$EPBF = \frac{O_2 \times 100}{PV - SIVC} = cc/min \qquad (22)$$

$$EPBF = \frac{O_2 \times 100}{PV - RA_{a.v.}} = cc/min \qquad (23)$$

Pulmonic stenosis, especially in cases of tetralogy of Fallot, may be accompanied by large collateral (bronchial) circulation or by a patent ductus arteriosus In such cases, total pulmonary blood flow or pulmonary capillary blood flow is larger than the main pulmonary arterial blood flow The former can only be calculated by indirect means, and the result is only a rough estimate

CHAPTER FOURTEEN

Summaries of Catheterization

TABLE VIII *Catheterization in Simple Shunts*

Disease	Pressures							Other significant data of catheterization	Other tests which may be useful	Remarks
	RA	RV	PA	PA wedge	LA	LV	AO			
Atrial Septal Defect or Anomalous Venous Return	Normal or slightly elevated (not above 10 mm)	Normal or slightly elevated (usually not above 50 mm)	Normal or slightly elevated (not above 50 mm)	Normal	Normal	Normal	Normal	Higher oxygen content in RA than in the average of SVC + IVC (>1 5 vol %)	ECG, Phono chest films (selective angiocardiography during catheterization for pulmonary anomalous venous return)	If RA pressure is markedly elevated there are additional lesions causing RV failure If the RV pressure is above 50 mm, significant pulmonary arteriosclerosis is present
Ventricular Septal Defect	Normal	Normal or slightly elevated (not above 60 mm)	Normal or slightly elevated (not above 60 mm)	Normal	Normal	Normal	Normal	Higher oxygen content is RV than in RA (>1 0 vol %)	Phono ECG chest films	If RV is markedly elevated there are additional lesions (pulmonic stenosis overriding aorta pulmonary arteriosclerosis)
Patent Ductus Arteriosus	Normal	Normal or slightly elevated	Normal or slightly elevated	Normal	Normal	Moderate increase of systolic pressure	Moderate decrease of diastolic pressure	Higher oxygen content in PA than in RV and RA (>1 0 vol %)	Phono ECG chest films	If RV and PA reveal hypertension there may be a reversed shunt (right to left) caused by pulmonary arteriosclerosis severe aortic stenosis or coarctation of the aorta

TABLE IX Catheterization in Complex Congenital Malformation

Disease	Pressures: RA	RV	PA	PA wedge	LA	LV	AO	[illegible]	[illegible]	Remarks
Tetralogy of Fallot	Normal	Markedly elevated	Normal or low	Normal	Normal	Normal	Normal	Low O_2 saturation of arterial blood (esp after exertion)	Angiocardiography ECG chest films Phono	
Eisenmenger Complex	Normal	Markedly elevated (similar to LV)	Markedly elevated (systolic pressure is as high as systolic of RV)	Normal	Normal	Normal	Normal	Low O_2 saturation of arterial blood (esp after exertion)	Angiocardiography ECG chest films Phono	
Complete Transposition of Large Vessels	Normal or slightly elevated	Elevated (systolic pressure is regulated by resistance in greater circulation)	Systolic is as high as that of LV	Normal	Normal	Low or Normal (systolic can be as high as that of RV)	Systolic is as high as that of RV	Low O_2 saturation of arterial blood	Angiocardiography ECG chest films	An atrial ventricular or ductal opening is usually present with equal and bidirectional shunts
Tricuspid Atresia plus shunts	Elevated	—	—	—	Normal or slightly elevated	Normal	Normal	Low O_2 saturation of arterial blood	Angiocardiography ECG chest films	In certain cases it is possible to pass the catheter through the mitral valve and a rudimentary right ventricle into the LA
Ebstein Malformation of Tricuspid Valve plus Atrial Septal Defect	Elevated during ventricular systole	Low or normal	Low or normal	Low or Normal	Normal or slightly elevated	Normal	Normal	Low O_2 saturation of arterial blood	Angiocardiography ECG chest films	

TABLE VIII *Catheterization in Simple Shunts*

Disease	Pressures							Other significant data of catheterization	Other tests which may be useful	Remarks
	RA	RV	PA	PA wedge	LA	LV	AO			
Atrial Septal De fect or Anoma lous Ven ous Return	Normal or slight ly eleva ted (not above 10 mm)	Normal or slightly elevated (usually not above 50 mm)	Normal or slightly elevated (not above 50 mm)	Normal	Normal	Normal	Normal	Higher oxy gen content in RA than in the aver age of SVC + IVC (>1 5 vol %)	ECG Phono chest films (se lective angio cardiography during cathe terization for pulmonary anomalous ve nous return)	If RA pressure is markedly elevated there are additional lesions causing RV failure If the RV pressure is above 50 mm , significant pul monary arteriosclero sis is present
Ventricu lar Septal Defect	Normal	Normal or slightly elevated (not above 60 mm)	Normal or slightly elevated (not above 60 mm)	*Normal*	*Normal*	*Normal*	*Normal*	Higher oxy gen content is RV than in RA (>1 0 vol %)	Phono ECG chest films	If RV is markedly elevated there are additional lesions (pulmonic stenosis overriding aorta pul monary arterioscle rosis)
Patent Ductus Arteriosus	Normal	Normal or slightly elevated	Normal or slightly elevated	Normal	Normal	Moderate increase of systolic pressure	Moderate decrease of dias tolic pres sure	Higher oxy gen content in PA than in RV and RA (>1 0 vol %)	Phono ECG chest films	If RV and PA reveal hypertension there may be a reversed shunt (right to left) caused by pulmonary arteriosclerosis se vere aortic stenosis or coarctation of the aorta

Pulmonic *Stenosis*	Normal	Elevated	Normal or *low*	Normal	*Normal*	Normal	Normal	—	*Phono* ECG chest films Angiocardiogram	There are three types valvular infundibular and combined If it is an infundibular stenosis a typical pattern and a low pressure are found in a separate chamber which is between the main cavity of the RV and the pulmonic valve If it is combined two gradients of pressure are found
Pulmonic *Insufficiency*	Normal	Normal or slightly elevated	Low diastolic pressure	Normal	Normal	Normal	Normal	—	EKY of PA and AO Phono ECG chest films	
Aortic Coarctation (*uncomplicated*)	Normal	Normal	Normal	*Normal*	Normal	Elevated	Elevated (above coarctation) decreased (below coarctation)	— —	Retrograde angioaortography, EKY of descending AO Phono ECG Pulse tracings	

TABLE X *Catheterization in Valvular Lesions or Coarctation*

Disease	Pressures							Other significant data of catheterization	Other test which may be useful	Remarks
	RA	RV	PA	PA wedge	LA	LV	AO			
Mitral Stenosis (with minimal or no insufficiency)	Normal	Elevated	Elevated	Elevated	Elevated	Normal (or low)	Normal (or low)	Low cardiac output or lack of *increase* with exertion	Phono ECG chest films	
Mitral Insufficiency (with minimal or no stenosis)	Normal	Normal or slightly elevated	Normal *or* slightly elevated	Normal *or* slightly elevated	Elevated during ventricular systole	Normal or slightly elevated	Normal	—	EKY of LA Phono, ECG, chest films	
Aortic Stenosis (with minimal or no insufficiency)	Normal	Normal	Normal	Normal	Normal	High	Normal or low	—	Phono EKY of LV and AO Pulse tracings (indirect and direct) ECG chest films	
Aortic Insufficiency (with minimal or no stenosis)	Normal	Normal	Normal	Normal	Normal	Elevated	Elevated systolic lowered diastolic	—	Phono EKY of LV and AO *Pulse tracings* (indirect and direct) ECG, chest films	
Tricuspid *Stenosis* (with minimal or no insufficiency)	High	Normal	Normal	Normal	Normal	Normal	Normal	—	Venous pressure jugular tracing	
Tricuspid Insufficiency (with minimal or no stenosis)	High during ventricular *systole*	Normal or slightly elevated	Normal	Normal	Normal	Normal	Normal	—	Venous pressure jugular and hepatic tracing ECG chest films	

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